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KERATITIS RAMIFICATA SUPERFICIALIS AND ITS CONNECTION WITH ASTHENOPIA, BLEPHAROSPASMUS NICTITANS AND PTERYGIUM

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JAVA

THE disease named above seems in the reality to be a new disease in so far as it has not been described in ophthalmic literature, although I described it myself in Geneeskundig Tijdschrift voor Nederlandsch-Indië in the year 1914. This is one of the reasons why I have waited so long a time before publishing it in ophthalmic literature. I wanted to make sure that it had not been mentioned there previously. If I am mistaken, I shall be thankful for any information.

In the first place I wish to emphasise, that this disease is quite different from the well known and well described keratitis ramificata or dendritica, as will be seen from the description in the following pages.

It seems nearly incredible, that I should have discovered a disease of the cornea unknown to other ophthalmologists and in a period where the cornea has been examined so thoroughly by means of slit-lamp and corneal-microscope, especially when I have to add, that the disease is very common in the tropics and in the summer time also in the temperate climates as for instance England and Denmark where I have also had the opportunity of

treating it. However, the greatest number of cases I have treated in Java.

The reason why it has formerly not been discovered is in the reality the perfection of the undulation of the cornea with secretion of the lacrymal glands. As known these glands are not effusing their content through a single big canal, but through 6-12 fine apertures on the posterior aspect of the upper eyelid. On account of this arrangement and of the perfect way in which the secretion is controlled we never see any flow of tears in the normal eye, although all surfaces are kept humid and shining.

This is evidently the effect of capillary action and diffusion. In no part of the eye is this action more important than to the cornea which only in this way can be kept shining and pellucid. The living normal cornea is therefore always covered with a layer of lacrymal fluid. It is so constant and so necessary, that we can say, physiologically speaking, that the cornea consists of six layers, viz., endothelium, membrana Descemeti, parenchyma, membrana Bowmani, epithelium and lacrymal layer.

This lacrymal layer has optically to fulfil two functions: (1) to keep the surface pellucid and (2) to give the surface a perfectly regular globular form. (1) needs no explanation, but how is (2) attained? In short we can say: through the combined laws of cohesion and adhesion. But I shall explain it more distinctly.

I beg to use an instance. When after an operation we pour the water or spirit out of a porcelain instrument tray, we see, that the remainder of the fluid does not form a regular thin cover on the bottom of the tray, but very quickly it forms separate drops.

The reason is, that the cohesion is stronger than the adhesion. The cohesion will cause water or spirit to form a globular body with perfectly regular, reflecting surface as we see it in the falling drop. In the instrument tray the drops are more flattened as a result of the gravity and adhesion, but yet they have a height of about half a millimetre. If the bottom under the drop should happen to have a small groove under one of the drops, 1/10 or 1/5 of a millimetre deep, this will not have any influence upon the surface of the drop which remains even and regular. In the same way the lacrymal layer on the cornea forms a regular, globular, reflecting surface, even if there should happen to be any very shallow depressions as in absence of some layers of epithelial cells or even of the whole epithelium.

This is the reason why the disease which I am going to describe in the following pages has been overlooked for so long a time.

In order to give a picture of the disease I shall describe a single case.

Mr. J. v. B., aged 50 years, pensioned official, Bandoeng, Java. Cons. January 28, 1936. For a long time much troubled in the

eyes. He cannot stand glare. The last two months blepharospasm of the left eye. Sometimes headache.

O.d. V. 5/36, with +20D. sph., 5/5, O.s. V. 5/36, with +20D. sph. 5/5.

B. has reading glasses d. and s. +3·5 sph.

Inspection of the eyes by good daylight straight inside a big window. With two fingers the right eye is kept widely open, and the patient is told to look along my right ear.

Very soon a pericorneal vascularisation takes place, and it increases quickly. The cornea is from the beginning perfectly clear and the surface all over bright and smooth as a mirror.

But suddenly a small groove is formed in the upper part. At the same moment the eye is getting irritated, and the cornea

FIG. 1.

FIG. 2.

would escape upwards under the upper eyelid, if I did not ask the patient to keep it still. The groove is extending and a new one is formed; but now the patient cannot stand it any longer, and the cornea disappears under the upper eyelid.

The left eye behaves in the same way, but I succeed in keeping it in position long enough to determine that the cornea is deprived of its epithelium in the upper part as shown in Fig. 1.

Now cocaine is instilled in both eyes, 1 per cent., 2 per cent. and 4 per cent., and the patient is again examined in the same way. Then after the eyes have been kept open 1 to 2 minutes, we find that the corneae are deprived of the epithelium in an extent as shown in the Figs. 1 and 2.

The hatched parts of the drawings here and in the following pages show the extent of the disease.

The reason why these epithelial defects did not appear from the beginning has been explained above.

The irrigation of cornea is so effective, that in many cases it will be necessary to stop or diminish the stream by means of cocaine in order to have a look at the uncovered cornea. In fact it will usually be necessary, if we want a full picture of the disease to drop 1 per cent. cocaine, 1 per cent. pantocaine and 4 per cent.

cocaine before the examination. If the upper eyelid be everted and kept well back from the cornea, the picture of the disease will often develop more quickly.

In fresh cases we find pictures as shown in Fig. 3 and Fig. 4; but usually the disease is of older standing, and the branches are then becoming confluent into flats with irregularly curved limits; but often we find at the same time isolated branches spread over the surface of the cornea.

FIG. 3.

FIG. 4.

Where the epithelium is missing the surface is slightly greyish and dull, in sharp contrast with the remaining shining surface of the cornea; but we never find any real dimness; as soon as the cornea is covered with its usual layer of tears, it is again perfectly clear, and the surface has regained its regular, globular form. Probably it is often only some single layers of epithelium that are missing, for sometimes I have found one part of the affected surface one layer deeper than the other.

The extension of the disease is very different as seen from the pictures in the following. In some cases I have found it spread over nearly the whole cornea.

Distribution

The disease is rather common, especially in the tropics where I have been working the last 30 years, at first 7 years in charge of a much frequented eye clinic in Semarang, Java, later on 15 years in the same city in charge of an eye hospital with 150 beds, and finally as a private practitioner.

Since I discovered the disease in 1908 I have treated a great number of cases here in Java.

Table II will show the number I have treated in the run of 15 years with a total of 3,335 and a yearly average of 222.

For the 11 years from 1921 till 1931 I have no statistics; but if we reckon the same average, we get a number of 2,442 for these 11 years. That would give a grand total of 5,777; thus if we reckon a grand total of 5,000, we may be on the safe side.

Also in northern countries we find this disease, especially in the warm season. During my stay in Europe in 1915 and 1922 I treated several cases in London and in different places in Denmark.

Aetiology

As to the aetiology the disease seems mainly to be due to climatic influences such as heat, glare, wind and dust. This will also explain why in the temperate climates it is mainly met with in the warm seasons. According to my experience it is not contagious. The racemose way in which it spreads, like growing, over the surface of the cornea, a circumstance which has caused me to call it " ramificata," seems to indicate some living agent; but there has not been any opportunity for bacteriological examination.

Symptomalogy

The symptoms of these patients are very different. The patients will complain about pain or itching in the eyes. They cannot stand the glare. They cannot stand work with the eyes, or at any rate only for a short time. This asthenopia is a very common symptom. Sometimes we find blepharospasmus nictitans, rather often cephalalgia, sometimes epiphora and photophobia. Vertigo is less frequent.

In Table I are given the statistics of four years showing the frequency of these different sufferings, and in the following pages they will be mentioned each singularly.

Asthenopia

In fact most cases of asthenopia are caused by keratitis ramificata superficialis, and when we make a habit of examining the cornea in the way I have described, we will find that the great number of cases of asthenopia nervosa will get reduced considerably.

That here we not have to do with an accidental coincidence, is proved by the fact that the asthenopia will disappear within one or two weeks, when the keratitis is well treated.

The asthenopia is felt and described by the patients in different ways. They will often say, that when they begin to work, they can see well, but after a period of half an hour, or longer, the vision is diminishing, and it is getting misty or blurred. The vision is sometimes diminishing so much, that the patients after a certain time cannot see any longer and have to stop work. Sometimes the letters are moving, when the patients try to read. Vision and refraction have of course always been examined and the last, when necessary, corrected.

Characteristic of these cases is the great contrast between the perfectly normal vision and the severe eye troubles of the patients. Table I shows the frequency of this symptom during four years. As seen, it is rather high, 38-45 per cent.

In this connection I shall mention another symptom which more intelligent patients will sometimes describe, viz., monocular diplopia of a special description seeing that the pictures are partly covering each other and that the one is more distinct than the

FIG 5.

other as shown in Fig. 5. I have myself been able to make this observation, when I was suffering from the disease. It comes into existence when the limit of the epithelial defect of the cornea is situated in front of the pupil, and the cornea then is partly getting dry.

Blepharospasmus Nictitans

Blepharospasmus nictitans is here not mentioned as a separate disease but as a symptom of the keratitis, because according to my 25 years' experience we will always find in these cases the keratitis, and when this has been cured, or already while it is improving, the blepharospasmus will disappear.

Formerly I considered this disease as a rather obstinate one; but after I found its origin, it was easily cured. I am here only speaking about the real, pronounced cases, and, as seen in Table I, these are not very common; but some spasm of the eyelid, coming into appearance by the examination, is often present.

TABLE I

YEAR.	Kerat. ram. superf.	Asthenopia	Bleph-spasm. nictit.	Cephal-algia.	Epiphora.	Photo-phobia.	Vertigo.
1932	283	128	2	24	4	–	–
1933	400	163	6	58	14	9	5
1934	381	149	8	64	11	3	3
1935	235	90	10	26	16	17	4

Cephalalgia

Cephalalgia is a rather common symptom as seen at the Table I, and it has not seldom been this symptom that has caused the patient to seek ophthalmic treatment, because it was combined

with some eye troubles. Usually it was evidently caused by the keratitis and disappeared with treatment.

It was present in 8·5-19·3 per cent. of the cases in Table I, and it was usually localised to the forehead.

Epiphora

Real epiphora was not often present, but very often the eyes of these patients watered more easily than normal eyes.

Photophobia

Real, pronounced photophobia was also rather rare; but usually the eyes were much more sensitive to light than normal eyes. This is of course a great hindrance in the examination and often makes the use of cocaine and pantocain necessary.

In some cases the photophobia has been the main symptom. I remember especially one case. The patient was a landowner from the mountains in Preanger in West Java. His photophobia was so strong, that it was perfectly impossible for him to control his grounds and his workers. He had been under the treatment of several colleagues for a long time, but without any improvement. He was just on the point of giving up his position, when he, as a last effort, came to me in Semarang in middle Java. It appeared to be a severe case of keratitis ramificata superficialis, and he consented to stay in Semarang about 5 weeks for treatment.

After the expiration of this period he was perfectly cured and could return home and take up his work.

Vertigo

As seen in Table I vertigo was not often present, and it was never in a heavy degree. That it was due to the keratitis appeared from the fact that it disappeared together with this disease.

Treatment

In the first place the eyes must be protected against glare, wind and dust. For this purpose the patients must wear dark spectacles, *when they are outdoors.* The glasses need not be too dark Crookes' B. or middle dark smoke are suitable. At the same time *they must keep as much as possible* indoors and for a time give up sport, especially swimming.

When suffering from asthenopia, they must give the eyes the necessary rest; thus when reading or writing they must stop work for some minutes or as long as necessary and then begin again. In very heavy cases only will it be necessary to stop work for some days, or longer. Smoke must be avoided. The patients

may well smoke tobacco outdoors, but may not stay in rooms with tobacco smoke.

For treatment at home fluorescin is the best remedy in the following solution: fluorescini milligram 400, carb. cal. 200, s. in sol. sublimati 1-10,000, grm. 20, d.s. eyedrops.

Fluorescein may not be applied seeing that it is somewhat irritating and has not the same therapeutic effect as fluorescin.

Seeing, that this remedy has a very strong staining effect, the patients must get special instructions about the use of it. A humid tampon must be ready on the table, the patient must turn the head well backwards, the eye is kept widely open with two fingers, and only one drop is put just on the middle of the eye. Then the tampon is put on top of the closed eye without rubbing. In this way the superfluous liquid will get absorbed by the wadding; otherwise it would stain the face. For social reasons this precaution is very important.

This instillation must take place 5 times a day. It is perfectly painless. When this treatment is continued for a long time a great improvement will usually take place; the most troublesome symptoms will diminish or disappear, and the patient will often be satisfied.

However the disease will not get fully cured in this way. For this purpose a stronger, local treatment will be necessary. Formerly I used sol. nitr. arg. 1 or 2 per cent. for brushing of the cornea or instillation of one drop only directly on the cornea after cocaine instillation.

After this treatment the patients would get rather severe pain against which I used dionine, seeing that cocaine is very hurtful in this disease. This treatment was repeated three times a week. The effect was rather good, but it was too troublesome for the patients. It was therefore a real relief for these patients as well as for myself, when I found, that antigonococcus serum also in these cases had the same effect as sol. nitr. arg. without causing any pain worth mentioning. The patients first get two instillations of 1 per cent. cocaine and then two instillations of the serum. This procedure is repeated three times a week for a period of 1-1½ months.

Sometimes a local anaphylaxis will develop after 2-3 weeks. The eyes will then get red and sore after the instillation and keep so some few days. The therapeutic effect is so much stronger, but still the serum treatment must be stopped for about a week and replaced by electrargol instillations. I have used this treatment 8-9 years.

The antigonococcus serum which I have used through all these years I have got from Parke, Davis and Co., Detroit, Michigan, U.S.A.

The firm informs me, that the serum is obtained from the blood of horses that have been treated with gradually increasing doses of live cultures of gonococcus. The activity of the serum is controlled by agglutination tests, so that a uniformly potent product is assured. Each lot is subjected to rigid bacteriological and physiological tests, both while in bulk and also after enclosure in the final container. The serum is served out in glass bulbs, closed by a rubber cap, each containing two c.c. The bulb must be opened aseptically, and the pipette that has to be used must also be sterilised.

A handy way to keep the bulb is to wrap it up in a slip of wadding, and put it into an ointment pot of 15 c.c. The pipette must have a point 33 mm. long, or the point must be bent so much that it can get to the last drop of serum, when the bulb is inclined. After the bulb has been opened, the serum will keep sound for one or two days in the tropics, and longer in a cooler climate.

Last year the same firm produced a new serum, called gonococcus antitoxin, containing a stronger antitoxin and less albumen. Dr. T. Anwijl-Davis has tried this new remedy in 157 different cases of gonorrhoeal infections with very promising results.

He describes these cases in the British Medical Journal, February 13, 1937, under the title of "Treatment of gonorrhoea with a specific antitoxin." According to the wish of Parke, Davis and Co., Detroit, I have tried it in the cases of eye diseases which I have hitherto treated with the old serum. It seems to have a similar effect; but it must be diluted with half its volume of distilled, sterile water, otherwise it is painful. The time has been too short to form a definite opinion, but my confidence is increasing.

Besides the serum treatment the patients use the fluorescin at home and wear a dark spectacle outdoors.

Usually the patients can continue their daily work. Only exceptionally they must have a short holiday of some few days or a week, very seldom longer.

In the final examination we may not use cocaine, seeing that it is hurtful for the corneal epithelium. I keep the eye widely open while counting slowly to 25. If the eye stands the glare well, and no epithelial defect appears, the patient is dismissed from the treatment in the eye clinic; but he must continue the treatment at home.

One month he must instil fluorescin four times a day and the second month twice a day. During these two months he must regularly wear the goggles outdoors but after this period only if he has to walk or drive a longer distance against glare and wind.

Case Histories

In the following I am going to describe some instances of patients suffering from this disease. In the accompanying drawings the hatched part will show, in how great extent the cornea was affected with the disease.

FIG. 6.

FIG. 7.

FIG. 8.

FIG. 9.

FIG. 10.

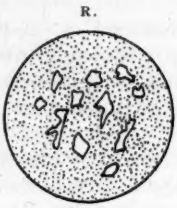

FIG 11.

FIG. 12.

FIG. 13.

FIG. 14.

FIG. 15.

FIG. 16.

FIG. 17.

FIG. 18.

FIG. 19.

FIG. 20.

FIG. 21.

FIG. 22.

FIG. 23.

FIG. 24.

FIG. 25.

FIG. 26.

FIG. 27.

FIG. 28.

FIG. 29.

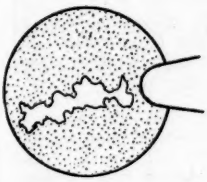

FIG. 30.

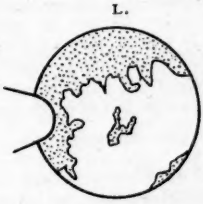

FIG. 31.

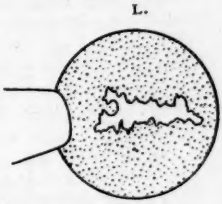

FIG. 32.

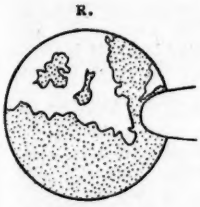

FIG. 33.

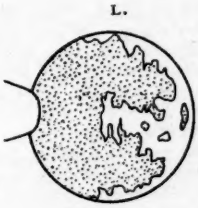

FIG. 34.

FIG. 35.

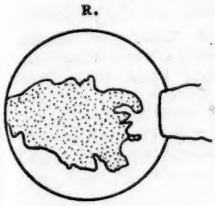

FIG. 36.

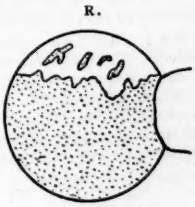

FIG. 37.

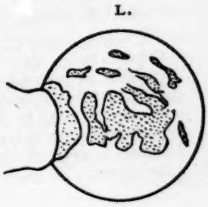

FIG. 38.

CASE 1.—Mr. Th. J. B., aged 30 years. July 11, 1927. Asthenopia, solar and for reading. Keratitis ramificata superficialis, both sides rather severe. Astigmatism. For about 10 years he had difficulties with his eyes. He has very straining eye work. He can only with difficulty perform his work and gets headache. He sleeps badly. He has suitable glasses, correcting his astigmatism. Vision with corr. o.d. 6/6, o.s. 6/6. Figs. 6 and 7 show the state of the corneae. August 10 he entered the eye-clinic and commenced treatment. Rp. fluorescin 5, Crookes' glasses B, 3 times a week brushing of the cornea with 2 per cent. sol. nitr. arg.

September 7, 1927. Formerly the patient was much troubled with headache, but the last two weeks it has perfectly disappeared. Sleep is more quiet than it used to be. He resumes his work, wearing dark spectacles outdoors and dropping into the eyes fluorescin 3 to 4 times a day.

November 4, 1927. Sometimes a little headache, when he has been working harder than usual, otherwise all right.

CASE 2.—Miss J. A. M., aged 34 years. October 19, 1927. Asthenopia. Ker. ram. superf. both eyes, rather severe. Is quickly tired by reading, and then she gets a sensation above the eyes, sometimes already at 1 o'clock in the afternoon, but usually not until the evening. Then she gets a strong feeling of sleepiness and must sometimes blink. The patient cannot stand strong light. R. emm. 6/6, L. emm. 6/6. When covering left eye she sees the letters double, the one picture partly covering the other and less distinct than this (Fig. 5). Fig. 8 will explain this phenomenon. As seen a branch of the disease is so near the centre of the cornea, that the edge of it will sometimes be in front of the pupil and will then break the rays of light in an irregular way. The usual treatment.

March 2, 1928. The weariness of the eyes has disappeared and there is no blinking any more.

CASE 3.—Mr. P. M., aged 44 years. April 23, 1928. During 2 months headache in the parietal region especially in the evening and night. The pain increases with reading, and when the patient has been reading a while, he gets sleepy. He cannot stand glare and is forced to wear dark spectacles.

R. emm. 6/6 }
L. emm. 6/6 } 0·45 in 45 ctm. distance.

With R. + 1·0 sph. }
L. + 1·0 sph. } 0·35 in 30 ctm. distance.

Patient gets a pair of spectacles with this correction. Figs. 9 and 10 show the state of the corneae. The usual treatment.

June 11, 1928. No headache any more. The eyes stand the glare quite well.

CASE 4.—Mr. I. J. E., aged 27 years. June 29, 1928. Asthenopia, Hyperm. Astigm. Ker. ram. both sides severe. When working in the evening he gets a pricking, burning pain in the eyes and then the vision sometimes gets blurred. He has a correct pair of spectacles and normal vision. The state of the corneae is shown in Figs. 11 and 12. He gets the usual treatment.

July 30, 1928. Patient has much less difficulty with his eyes. October 10, 1928. The eyes feel quite normal.

CASE 5.—Mrs. Gr., aged 63 years. April 6, 1933. Photophobia. Ker. ram. superf. both sides severe. Has much trouble from the glare. Extension and form of the disease is shown on the two Figs. 13 and 14. Rp. fluorescin x 5, dark spectacles, instillation of antigonococci-serum, Parke, Davis & Co., Detroit, every second day. May 4, 1933. Can stand the glare much better.

May 22, 1933. Has been away one week on account of disease.

May 30, 1933. The corneæ normal, no symptoms. Discharged from treatment.

CASE 6.—Mr. Ch. O. van der P., aged 43 years. June 19, 1934. Asthenopia, Cephalalgia, Ker. ram. s. severe in right eye, very severe in left. When the patient is getting tired, he cannot see well. He is suffering much from headache. The eyes are red. Figs. 15 and 16 show the state of the corneae, R. emm. 5/5, L. emm. 5/5, 0·35 in 30 ctm. He gets the usual treatment, but can only visit the clinic from time to time. Seeing that he is driving much in an open motor-car, he gets a pair of dark spectacles with side covering for this purpose.

July 16, 1934. Patient can see well. No trouble from the eyes any more, not even when on tour. There is still some keratitis.

October 5, 1934. No headache or trouble from the eyes which are perfectly cured.

CASE 7.—Raden N., aged 8 years, boy. May 29, 1935. Asthenopia severe, Ker. ram. s., both sides severe. The patient is nervous. At school, when writing the letters are at first good, but very soon they get very bad. His marks are bad for writing. Figs. 17 and 18 show the state of the corneæ. R. 5/5, L. 5/5, 0·35 in 30 ctm.

July 6, 1935. Patient is cured. The writing at school much better.

CASE 8.—Mrs. E. K., aged 35 years. October 23, 1935. Cephalalgia, Asthenopia Epiphora. Ker. ram. s. severe. Burning feeling in the eyes, they are watering. Often headache. When she is reading, the eyes are quickly tired. R. H. 0·5, 5/5, L. emm. 5/5, 0·35 in 30 ctm. The state of the corneae is shown in Figs. 19 and 20. She gets the usual treatment and is cured November 18, 1935.

CASE 9.—Mr. A. A. L. G., aged 57 years. November 29, 1935. Vertigo, Keratitis ram. s., severe both sides. Pteryg. incip. on each side. During 8 months the patient is suffering from giddiness and has a feeling of weariness in the eyes. Some twitching in the eyelids. Urine clear without albumen or sugar. The state of the corneae is shown in Figs. 21 and 22. Patient got the usual treatment.

November 28, 1935. The corneae are cured. The " heads " of the pterygia have withered. The giddiness has diminished very much and disappeared after a short time by the use of br. amm.

CASE 10.—Mr. J. J., aged 45 years. January 23, 1936. Asthenopia for reading. Keratit. ram. s. For 8 years he has difficulties with his eyes. When he has been reading 5 minutes, he cannot see anything any more. He has a pair of spectacles; but they do not help him. In the evening it is still worse. R. 6/8, L. 6/8. There is some little astigmatism, but the patient does not want any correction. He has suitable reading glasses. Figs. 23 and 24 show the condition of the corneae. He gets the usual treatment. The condition of the eyes is improving rapidly, and March 18, 1935, he is dismissed as cured.

CASE 11.—Mr. J. B., aged 55 years. January 28, 1936. Hypermetr. Presbyopia, Blepharospasm nictitans left eye. Asthenopia, Ker. ram. s. severe both sides. The patient has much trouble from his eyes and cannot stand the glare. The last two months he has suffered from blepharospasmus nictitans of the left eye. He often has headache. He cannot stand his office work. R. 5/36, with + 2·0 D. sph. 5/5, L. 5/36, with + 2·0 D. sph. 5/5. He gets this correction with Crookes' B. For reading he has + 3·5 D. sph. for both eyes. V. 0·35 in 30 ctm. The patient must stop his office work. He gets the usual treatment. Figs. 25 and 26 show the state of the corneae.

February 8, 1936. The blepharospasm has diminished very much.

February 29, 1936. Much improved. There is still a little keratitis.

March 4, 1936. To-day he has done his office work without any difficulty. No blepharospasm any more.

March 19, 1936. The keratitis is cured. He feels quite well.

CASE 12.—Miss W. J. S., aged 20 years. November 20, 1936. Cephalalgia, photophobia, Ker. ram. sup. both sides, severe, scotoma scintillans. When working (typing) patient gets headache, and she cannot stand the glare. Rather often scotoma scintillans. The state of the corneae is shown in the Figs. 27 and 28. The usual treatment.

November 30, 1936. No headache any more.

December 11, 1936. The patient states spontaneously that she has no trouble with her eyes any more.

December 16, 1936. Discharged from medical treatment.

CASE 13.—Mr. L., aged 35 years. November 30, 1936. Photophobia. Cephalalgia. Ker. ram. sup. severe. Cannot at all stand the glare and wears dark spectacles. Is suffering from headache in the forehead. A feeling of pressure in the eyes. When looking at the test types on the wall with left eye only, he sees a double picture as a shadow, partly covering the original. When looking at Fig. 29, we see, that the border of the epithelial defect is just passing

the middle of the pupil. That explains the double picture. The usual treatment.
December 12, 1936. The patient communicates, that the headache has ceased some few days ago.
December 30, 1936. The patient can stand the glare much better.
January 12, 1937. Feels well. Clinical treatment finished.

Pterygium

The number of cases of this disease we will find in the Tables II and III, but the last gives only the cases that have been operated upon.

The reason why it is mentioned here is, that there seems to be a strong connection between the two diseases. After my attention had been fixed upon this matter in the year 1918, I have examined the cornea in the way described above in all cases of pterygium. That means in the years 1919, 1920, 1932, 1933, 1934 and 1935, 172 cases. From 1921 and 1922 I have no statistics and for the years 1923-1930 both inclusive, I have only the operated cases, altogether 73. This makes a total number of 245.

In all these cases without one single exception I have found on the cornea of the affected eye keratitis ramificata superficialis in a heavy degree.

And in nearly all cases with some very few exceptions only the pterygium was in touch with the affected part of the cornea.

TABLE II

YEAR.	Kerat. ramif. superf.	Pterygium.
1909	207	22
1910	250	22
1911	164	29
1912	44	25
1913	154	33
1915	166	13
1916	241	22
1917	208	29
1918	225	38
1919	179	51
1920	197	71
1932	283	8
1933	400	12
1934	381	15
1935	235	15
Total	3,334	405

TABLE III

YEAR.	Pterygium
1923	11
1924	8
1925	9
1926	15
1927	14
1928	1
1929	7
1930	8
Total	73

The figures comprise operated cases only.

When the keratitis was treated in the way described above I found that after a time the " head " of the pterygium was withering away, and it stopped growing.

Several small pterygia were cured in this way without operation.

At the same time I observed that an effective treatment of the keratitis was the best way to prevent relapse of the pterygium.

These different facts give the impression, that the pterygium is caused by the keratitis, and that we here in reality have to do with a sort of natural healing in that the conjunctiva bulbi is trying to cover the denuded parts of the cornea, but—alas —not always with the best result.

The cure consisted in the usual operation combined with the treatment of the keratitis. The very small pterygia only were cured without operation.

In the following I am going to describe some cases of pterygium. The figures show the distribution of the keratitis in the same way as in the other diagrams.

CASE 1.— O. K. H. April 4, 1923. Pterygium. Ker. ram. super., severe. Figs. 30 and 31 show the state of the corneæ. He gets instillation of sol. nitrat. arg. 1 per cent., fluorescin, and protargol.

April 19, 1923. Op. pterygii. Left.

June 9, 1923. Op. pterygii. Right. After a treatment of about three months he was perfectly cured.

CASE 2.—Mr. A. J., aged 28 years. October 26, 1923. Small recurrent pteryg. on right side, medium sized on left. Ker. ram. superf. severe on right and worse on left side. The state of the left cornea is shown in Fig. 32. He was treated with instill. of sol. nitr. arg. 1 per cent., fluor. and prot. 20 per cent.

November 6, 1923. Oper. pteryg. Left eye. When he went home after about one month of treatment, he was nearly cured.

CASE 3.—S. H. P., Javanese, aged 25 years. October 15, 1925. Ker. ram. s. severe both sides, double pterygium. The state of the corneae is shown in Figs. 33 and 34. Rp. fluor. x 4; dark spectacles, coc. instill. sol. nitr. arg. 1 per cent. protargol 20 per cent., dionine.

October 8, 1925. Oper. pteryg. Right eye. The left eye was not operated upon. The small pterygium withered away during the medical treatment of the cornea.

October 21, 1925. Cured, going home.

CASE 4.—F. S. H., aged 49 years. August 9, 1926. Asthenopia. Pterygium both eyes. Keratitis ramificata superf. very severe both sides. R. Emm. 5/6, L. Emm. 5/6. The state of the right cornea is shown in Fig. 35. Rp. coc. 2 per cent. x 2, instill. nitr. arg. 1 per cent., protargol 20 per cent. and later on coc. 1 per cent. x 2, instill. of antigonococcic serum. Dark spectacles.

September 2, 1926. Oper. pteryg. R. eye. September 9, 1926. Oper. pteryg. L. eye. October 5, 1926. Cured. Going home. Cont. fluor. and dark spectacles 2 months.

CASE 5.—Mr. F. B. W., aged 43 years. October 3, 1927. Asthenopia severe. Pterygium R. side. Ker. ram. severe both sides. Malaria chron. The state of the right cornea is shown in Fig. 36. When the patient in the evening is working, the eyes get hot, and begin to water. It has been like that for about a year. He has R. & L. 1·0 D. spher. He cannot stand the glare, especially on the lime-covered roads. The spleen reaches the curvature. Four years ago he had a fit of tertian malaria. Rp. decoctum foliarum Blumeae balsamiferae No. 7 ad remanentiam colaturae 750 c.c. The whole lot must be taken in the run of a day and a night. Fluor. x 4, dark spectacles, instillation of serum antigon.

October 10, 1927. The spleen is normal in size (12·5 ctm. shorter than October 3, 1927. Cont. decoct. fol. Blum. bals., 6 leaves. Patient feels much better.

October 17, 1927. Patient stands the glare much better.

October 31, 1927. Formerly he would get headache if he had to work in the evening, but at present none. His appetite has increased very much. The eyes stand examination much better.

November 15, 1927. Fully cured. The pterygium has atrophied.

CASE 6.—Mr. T. I. D., aged 27 years. June 25, 1928. Pterygium both eyes. Ker. ram. sup. severe both sides. The state of the corneae is shown in Figs. 37 and 38. R. V 5/5, L. V 5/5. Patient is suffering from chronic malaria and is treated with quinine. The spleen is a little enlarged. Rp. instillation of antigon. serum, dark spectacles, fluor. x 3.

July 10, 1928. Oper. pteryg. R. eye.

July 17, 1928. Oper. pteryg. L. eye.

August 5, 1928. Cured. Discharged from treatment.

Conjunctivitis Aestivalis

The reason why this disease is mentioned in this connection is that it is often found together with ker. ram. sup.

Table IV will show how often this combination has taken place in the years 1932-1935. But still there is a much greater amount of keratitis without conj. aest., and on the other side there is a certain number of cases of conj. aestiv. without keratitis. Under these circumstances it seems to be most rational to consider the two diseases as being independent of each other. The frequent coincidence can be explained by the partly common aetiology, viz., glare, heat and dust. On one point however there is a decided difference in the aetiology seeing that the conjunctivitis will only develop when there is a deficiency in the calcium metabolism and the production of parathyroidine.

Consequently these two substances have a great curative effect in this disease, the first especially in the form of calcium phosphate.

TABLE IV

YEAR.	Kerat. ram. s. only.	Conjunct. aestival. and ker. ram. s.	Conjunct. aestival. only.
1932	185	98	8
1933	292	108	8
1934	273	108	9
1935	182	53	10

Summary

The author has formerly only once published a description of the eye disease here described in a medical periodical in the Dutch East Indies in the year 1914.

In ophthalmological literature it has never been described either by the author, or by anyone else, as far as is known.

It is exclusively a disease of the epithelium. In fresh cases we find a branch formed figure on the surface of the cornea, as if ..lad been carved out with a small hollow chisel. By and by the ..gures join up and in this way arise more or less extensive epithelial defects.

The reason why the disease has so long avoided discovery is, that the living cornea is always covered with a thin layer of lacrymal fluid which layer according to the laws of coherence and adherence will always have a globular form and a regularly reflecting surface just like a drop of water. For examination it will consequently be necessary to keep the eye of the patient widely open until this lacrymal layer has evaporated. For this purpose it will often be necessary to instil cocaine, or still better pantocain repeatedly previously to the examination.

The description of the treatment must be read *in extenso*.

Special attention is given to the connection between the described eye disease and pterygium. Seeing, that the author since he 20 years ago began this examination has found a high degree of keratitis in all cases of pterygium, and seeing, that the head of the pterygium will always wither away, when the keratitis is well treated, he comes to the conclusion, that the pterygium is caused by the keratitis.

The connection between the keratitis and conjunctivitis aestivalis is also considered. The conclusion of the author is, that they are two separate diseases.

The fact that they often are found together, is explained by the fact, that they have a partly common aetiology.

INTRA-OCULAR TUMOURS

BY

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IN practice, intra-ocular tumours are vaguely classified as gliomata if they originate in the retina, or as melanomata if they have originated in the choroid and contain brown pigment that does not give the iron reaction. Those attempting greater precision place most of the gliomata in the more restricted group of retinoblastomata. The problem, however, is not so simple, for on closer examination a more detailed grouping becomes necessary.

Tumours occurring in other parts of the body have been classified with varying degrees of success, according to the development of the tissue of the part. Similarly, the classification of intra-ocular tumours can be based on retinal and choroidal development. In the present paper a series of 60 intra-ocular tumours is reviewed and a classification is attempted on a developmental basis.

Material

In the earlier stages the eyes were fixed in 10 per cent. formol saline, frozen with carbon dioxide snow, cut antero-posteriorly through the optic nerve, and then prepared according to the paraffin method. Later Helly's fluid was employed as the fixative, and the eyes were cut after fixation as for the previous method. By this latter procedure the inner portions of the eye did not collapse since the vitreous had coagulated during fixation. The sections were stained with haemalum and eosin, and these stains proved adequate in most cases.

According to some workers all neurological material must be prepared by complicated stains and impregnation methods. However, the haemalum and eosin appearance of the various cells in neural development have been described repeatedly, and although

the fine details may be lacking, haemalum and eosin staining gives sufficient detail for diagnostic purposes and it is better adapted to routine examinations than the more complicated methods.

Existing Classifications

Collins and Mayou (1925) group intra-ocular tumours as a whole into the following divisions:—

1. Teleplasms, (a) mesoblastic (choroid),
   (1) pigmented,
   (2) non-pigmented,
   (b) neural epiblastic,
   (c) of mixed origin.
2. Metastatic growths.
3. Cysts.

In spite of this attempt to gather all tumours under one scheme of classification, the general tendency is to treat the retinal and the choroidal groups separately. Thus Grinker (1932) divides the retinal tumours into:—

1. those arising from the primitive retinal or ciliary epithelium, medullo-epitheliomata;
2. those arising from the hypothetical bipotential retinoblast, retinoblastomata;
3. and those having their origin in primitive spongioblasts or neuro-epithelium, neuro-epitheliomata.

Tumours of the choroid have been classified by Callender (1931) into 4 main groups, all of which are considered melanotic tumours.

1. Spindle cell type (a) heavily pigmented,
   (b) slightly pigmented; prominent nucleolus.
2. Epithelioid type—pigment variable.
3. Fascicular type—pigment scanty; cells in columns and fasciculi.
4. Mixed type—heavily pigmented; commonest.

These classifications do not take into consideration several important points, the chief of which are:—

(1) that retinal tumours, because of their neural origin, should be classified on a basis similar to that adopted for the glial tumours of the central nervous system, and (2) that choroidal tumours need not invariably be of the melanotic type.

Neural Cell Development

Tumours of the retina and the optic nerve arise from the neural elements of those structures or from their blood vessels. As tumours of neural origin are by far the commoner it will be of advantage to consider in a general way the development of the eye. In embryonic development the retina and the optic nerve originate from an evagination of the primitive optic vesicle which is made up of primitive medullary epithelium of the type found in the neural tube. Progressive development of these cells eventually gives rise to the neural elements of the adult retina. The various stages are shown in the following diagram given by Grinker (1932).

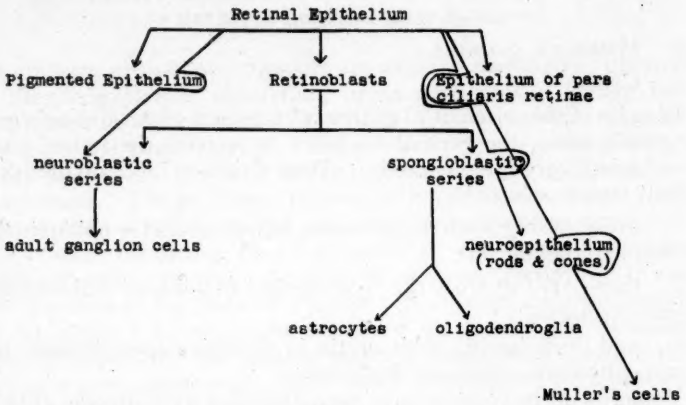

FIG. 1.

As the retinoblast is considered to be practically identical with the medulloblast of the central nervous system, it becomes evident that all the types of cells utilised in the classification of the neural tumours of the central nervous system are present in the development of the retina. This emphasises the possibility of an analogous classification for tumours of the retina.

In practice, it is as a rule impossible to classify the tumour cells on as detailed a basis as shown in Fig. 1. For the purpose of constructing a workable classification a more simplified scheme of development will be useful.

Broadly considered cells of neural origin pass from the neuroepithelial to the spongioblastic stage; the latter can be divided into (1) a primitive or apolar spongioblastic and (2) a polar spongioblastic group. From this point the more advanced specialisation becomes evident, and in the case of the elements

other than the glia the next phase is the neuroblastic which is followed by the neurocytic phase. In a corresponding way the glia would at these stages go through both the " blast " and the " cyte " stages. The abbreviated scheme of development is graphically represented in Fig. 2.

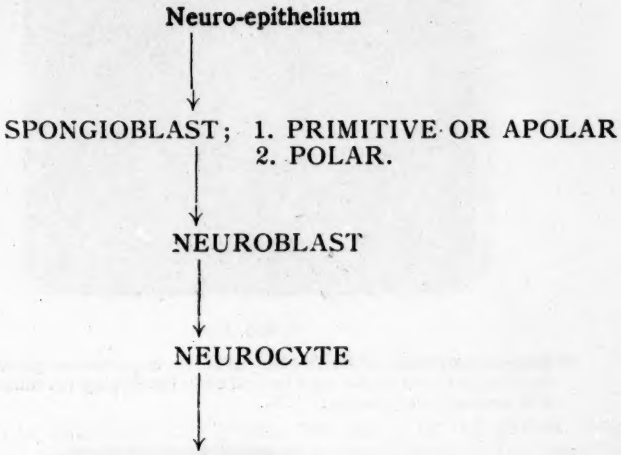

FIG. 2.

Classification of Tumours of the Retina

In any classification simplicity and elasticity are important. In order to achieve this only the most outstanding features of the tumours were utilised in constructing the classification. On this basis only 3 major types were necessary, namely, the neural tumours, the sarcomata and the melanomata of the choroid.

The neural tumours were divided into the following groups on the developmental basis, and from the less differentiated to the more differentiated types.

(*a*) *Neuro-epitheliomata.*—This group includes the medullo-epitheliomata and the neuro-epitheliomata of Grinker's classification. The tumours of this group may be composed essentially of columnar epithelium (Fig. 3) resembling primitive medullary and retinal epithelium, and showing evidence of differentiation into retinoblasts, neuro-epithelium with rosette formation (Fig. 4), spongioblasts, ependymal cells (Fig. 5), and even adult glia. In some tumours there are features of the embryonic retina. Mitotic figures may be quite numerous.

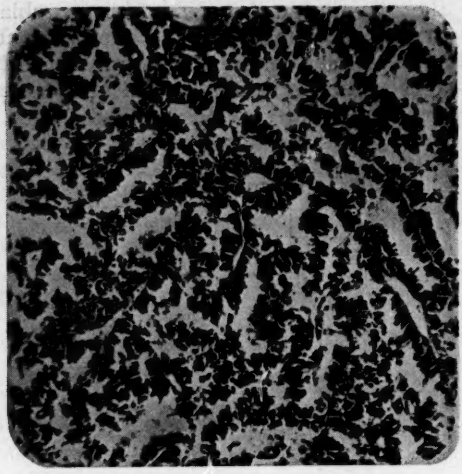

FIG. 3

Neuro-epithelioma of the Retina. There are numerous spaces lined by columnar cells of the type seen in early developing nervous tissue. The stroma is very scanty. ×80.

FIG. 4.

Neuro-epithelioma of the Retina. This shows continuity between the tumour and the retina. Rosettes are numerous. Such tumours have been referred to as adenomata. ×80.

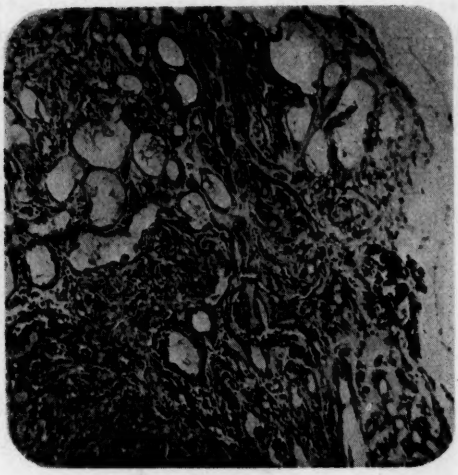

FIG. 5.

Neuro-epithelioma of the Iris. The numerous spaces are lined by flattened ependymal cells. ×80.

Many tumours of this group are made up of rather large spheroidal and polyhedral cells of the type seen in the so-called neurogenic sarcomata of the peripheral nerves. The cells as a rule have an eosinophilic cytoplasm and a distinct and well stained nucleus. This type is probably very undifferentiated and even more so than those of the previous types.

(*b*) *Spongioblastic Group.*—1. *Apolar or Primitive Spongioblastomata.*—The tumour classed under this heading is generally termed a retinoblastoma, and it is very similar to the medulloblastoma of the central nervous system. According to Penfield the medulloblast is an apolar spongioblast and as the medulloblast and the retinoblast are identical, the retinoblast can also be considered an apolar spongioblast. Hence in order to retain continuity in the classification the retinoblastoma may be termed an apolar or primitive spongioblastoma.

The growth is made up of small or polyhedral cells, some of which will show tapering at one end; the cells as a whole have scanty cytoplasm. They are closely packed with large, darkly staining nuclei (Figs. 6 and 7). Mitotic figures are numerous. The tumour may also contain pseudo-rosettes, polar spongioblasts, astroblasts, and astrocytes.

The tumour usually originates in the posterior part of the retina and even from the papilla. It may infiltrate and produce metastases.

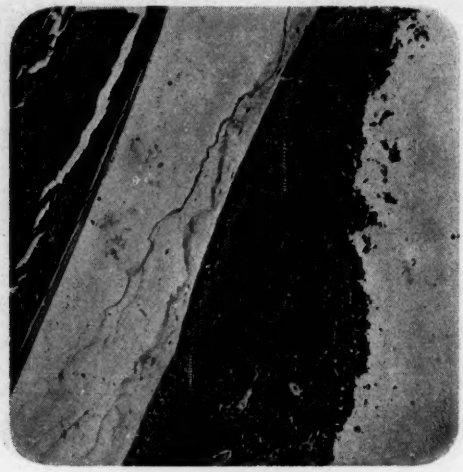

FIG. 6.

Apolar Spongioblastoma or Retinoblastoma of the Retina. Its relationship with the retina is distinctly seen. The tumour is made up of small closely packed cells with darkly staining nuclei. ×50.

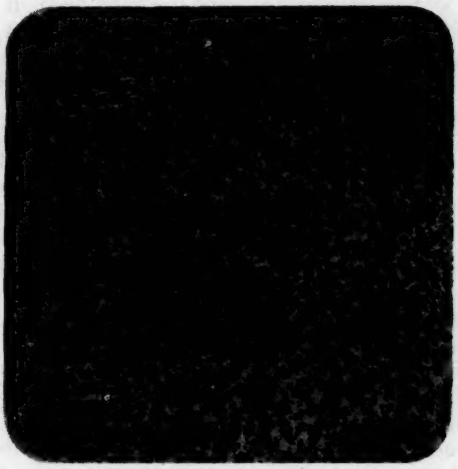

FIG. 7.

Apolar Spongioblastoma or Retinoblastoma of the Retina. A high-power view of the same tumour to show the closely packed small cells with occasional tapered cells amongst them. ×125.

2. *Polar Spongioblastomata.*—As in the central nervous system, the gross histological features of the polar spongioblastoma suggest a close similarity with the spindle cell sarcoma of other tissues. If a tumour of the eye gives the impression, histologically, of a spindle cell sarcoma, spongioblastoma polare must be eliminated first.

The tumour is made up uniformly of fairly large fusiform cells (Fig. 8) with a cytoplasmic process from one or both ends. These processes are not connected with the connective tissue septa, nor

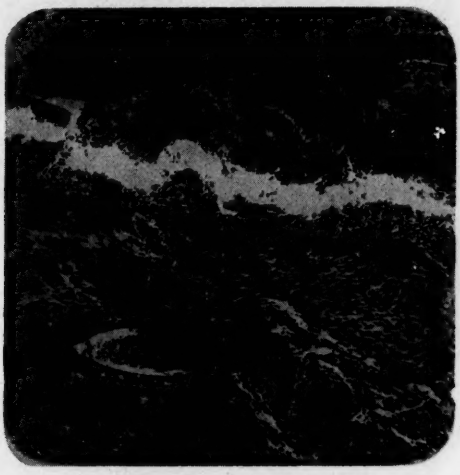

FIG. 8.

Polar Spongioblastoma. Fusiform cells are abundant and typical of this type of neoplasm. ×50.

attached to the blood vessels, and they do not stain specifically with Mallory's phosphotungstic acid stain. In places the cells tend to be parallel and at times closely resemble the central neurinoma. As a rule, the blood vessels of the tumour are well formed. As the tumour is slowly growing, mitotic figures are rare and necrosis is not common.

These neoplasms are generally included in the melanoma group.

(*c*) *Neuroblastic Group.*—Tumours composed of neuroblasts or neurocytes can be distinguished both by the advanced degree of differentiation evident in the tumour, in the form of attempts to develop recognisable retinal structures (Figs. 9 and 10), and also by the presence of a deeply staining nucleolus in a well-defined

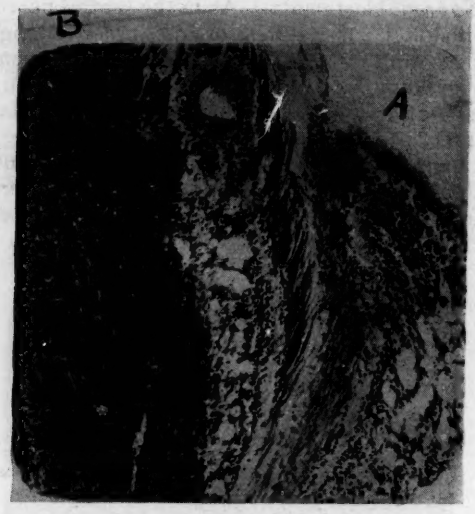

FIG. 9.

Neuroblastoma of the Retina. One half of the tumour shows retinal differentiation of an advanced type (A) but in the portion marked (B) neuroblasts predominate. ×50.

FIG. 10.

Neurocytoma of the Retina. The neoplastic process has resulted in forming recognisable retina with the formation of layers or zones as seen in the retina. ×80.

nucleus. The predominance in the growth either of neurocytes or neuroblasts, as the case may be, determines whether the tumour is to be classed as a neurocytoma or a neuroblastoma.

Although only the pure types have been described throughout, the fact remains that mixed and intermediate types are very

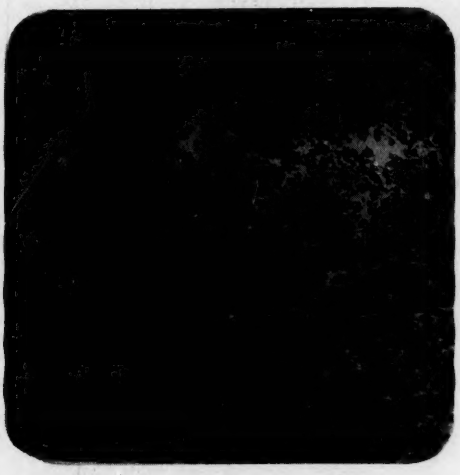

FIG. 11.

Polar Spongioblastoma showing Neuro-epithelial Elements. ×125.

common, as for example in *Fig. 11* where polar spongioblasts are common but neuro-epithlial features are also present. These mixed types have formed connecting links between all the groups described in the above classification.

Results—Retinal Tumours

In the following table the retinal tumours are grouped on the basis explained above, and along with this are given details relevant to this investigation.

Classification of Tumours of the Choroid.—Tumours of the choroid have their own special features and these must be considered in dealing with the classification of tumours of this region. In addition, the structural peculiarities of the choroid itself must be taken into consideration. Since the choroid forms the outer sheath to the retina, some tumours arising in that region might show some similarity with the perineural fibroblastomata.

TABLE I

Retinal Tumours Classified on a *Developmental Basis.*

No.	Sex and Age	Site	Pigment	Benign or Malignant
NEURO-EPITHELIOMA				
28-115	—	Retina	4+	M.
28-346	Male, 6/12	Retina	1+	M.
33-172	*Male, 39*	Iris	2+	M.
29-400	Male	Retina	—	M.
31-455	*Male*	Retina	1+	M.
34-248	Male	Retina	—	M.
35-226	*Male, 59*	Retina	2+	M.
APOLAR (PRIMITIVE) SPONGIOBLASTOMA				
28-161	*Male*	Retina	4+	M.
25-385	Female	Retina	1+	M.
28-288	—	Retina	1+	M.
25-732	Male	Retina	1+	M.
28-381	*Male, 3*	Retina	+	M.
27-118	—	Retina	—	M.
27-323	*Male*	Retina	—	M.
28-344	Male, 3	Retina	—	M.
34-91	Male, 2	Retina	1+	M.
34-144	Female, 7	Retina	—	M.
34-335	Male, 2	Retina	—	M.
36-180	Female	Retina	—	M.
37-209	Male, 4	Retina	—	M.
POLAR SPONGIOBLASTOMA				
27-232	*Male, 45*	Retina	1+	
28-348	—	Retina	1+	
31-144	Female	Choroid	1+	
31-230	Male	Choroid	+	
33-650	Male	Choroid	4+	
34-357	Male, 36	Retina	—	
34-474	Male	Retina	1+	? B.
35-69	Male	Choroid	3+	
35-418	Female	Retina	1+	
36-246	Female, 49	Choroid	2+	
37-118	Male, 79	Ciliary body	1+	
37-397	Male, 37	Retina	4+	
37-410	Female, 44	Choroid	4+	
NEUROBLASTOMA				
31-260	Male	Retina	1+	M.
34-293	Female	Retina	—	M.
35-576	Male, 64	Retina	1+	M.
NEUROCYTOMA				
32-52	Male, 8	Retina	1+	M.
33-681	Male, 3	Retina	—	B.

Furthermore, the particular differentiation of the choroid itself gives rise to neoplastic features of a distinctive type other than the presence of melanin, for melanin alone is of doubtful value as a basic feature for the classification of tumours of the choroid. Since a great variety of disturbances of the choroid will give rise to an increased production of choroidal pigment, the presence or absence of pigment in the tumour takes on a secondary significance. Hence the presence of pigment in an intra-ocular tumour

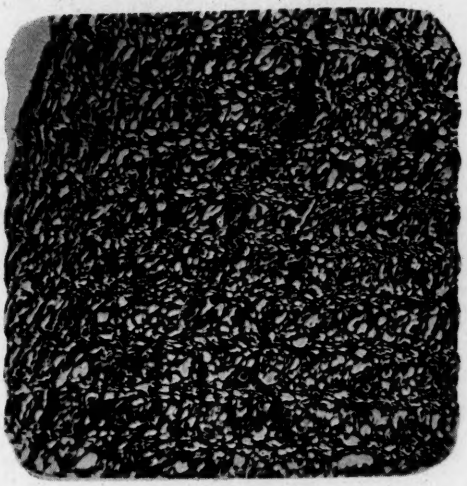

FIG. 12.

Sarcoma of the Choroid. This shows the differentiation of the choroidal tumour into the cavernous form. The stroma is made up of small fusiform cells as seen in the choroid. ×80.

does not imply that the tumour is primarily one of the choroid; neither can it be inferred on this ground alone that the tumour is of melanotic origin. The presence of melanotic pigment in a tumour merely implies that the growth has disturbed the choroid. The classification of choroidal tumours can be based with more certainty on cell differentiation rather than on the presence or absence of pigment. If the tumour is of choroidal origin the cellular differentiation should tend towards the choroidal structure, and in its more advanced form it should tend to show the cavernous appearance of the choroid (Fig. 12). In their malignant forms these tumours are sarcomata.

The malignant tumours of the choroid can be divided into 3

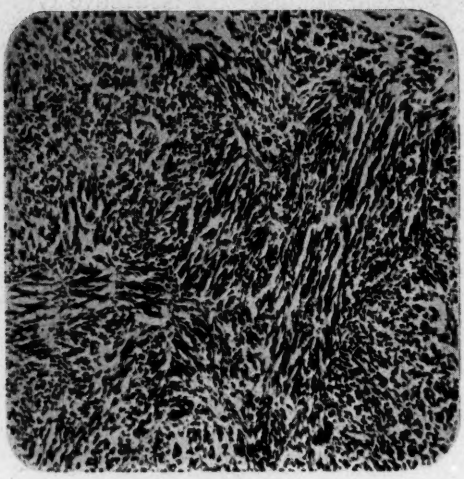

FIG. 13.

Sarcoma of the Choroid. In the malignant portion of the tumour which is included in this photograph, the cells are of the spindle cell type. In other parts cavernous structures were evident. ×80.

types: (*a*) the round cell sarcoma, (*b*) the spindle cell sarcoma (Fig. 13), and (*c*) the more specialised types such as the myxosarcoma. In all these types there are as rule areas showing the fenestrated or cavernous appearance of the choroid. In other words the tumours are essentially malignant choroidomata.

Results—Tumours of the Choroid

(1) *Sarcomata.*

TABLE II

Sarcomata of the Choroid divided into Groups according to Cell Types.

ROUND CELL TYPE

No.	Sex and Age	Site	Pigment	Benign or Malignant
28–93	Female	Choroid	+	M.
34–8	Male	Choroid	1+	M.
35–443	Male	Choroid	1+	M.

TABLE II—*continued*

No.	Sex and Age	Site	Pigment	Benign or Malignant
SPINDLE CELL TYPE				
27–200	Male, 52	Choroid	3+	M.
28–83	Male, 60	Choroid	2+	M.
35–54	Female, 70	Choroid	1+	M.
33–48	Male	Choroid	3+	M.
33–285	Female, 37	Choroid	4+	M.
33–686	Female, 51	Choroid	5+	M.
34–21	Male, 35	Choroid	1+	M.
34–181	Male, 58	Choroid	1+	M.
34–405	Female	Choroid	3+	M.
34–476	Female, 63	Choroid	4+	M.
36–129	Female	Choroid	1+	M.
37–167	Male, 55	Choroid	3+	M.
37–261	Male	Ciliary body	2+	M.
37–470	Male, 37	Choroid	2+	M.
MIXED TYPES				
32–564	Male	Choroid	1+	M. myxosarcoma

(2) *Melanomata.*—The remaining group is made up of the melanomata. These are tumours of the choroid and are highly pigmented as a rule. The tumour cells are of the large triangular or branching type; in parts they may be of the fusiform, rounded or polyhedral types. As the presence of melanotic pigment alone is an inadequate criterion, the fundamental feature of this group is the presence of the branching melanotic cells even though these may form a small part of the tumour.

TABLE III

Melanomata of Choroid.

No.	Sex and Age	Site	Pigment	Benign or Malignant
28–134	Male	Choroid	4+	M.
32–247	Male	Choroid	2+	M.
37–168	Male, 67	Choroid	5+	M.
37–197	Male, 60	Choroid	5+	M.

Discussion

The findings are limited because clinical correlation could not be established. Information as to age was lacking in many cases, and this further limited the scope of this investigation. In spite of these deficiences the histological features of the tumours are

sufficiently distinct, and their histogenesis sufficiently clear to permit them to be divided into a number of groups according to the main constituent cell type of the tumour.

The classification of tumours of the retina hardly requires any further discussion. The present scheme has a developmental basis and the criteria for each group are sufficiently general to allow as much latitude as is the case in the classification of the glial tumours of the central nervous system. These points have already been dealt with in some detail.

Of the neural tumours 7 fell into the neuro-epithelial group and all of these were malignant. Only 1 appeared to originate in the iris. Two out of the 7 were devoid of pigment, while the remainder contained varying amounts of melanin. The apolar spongioblastoma or retinoblastoma group was made up of 12 cases and these, too, were all malignant. The age was available in 5 cases only; 4 were either 2 or 3 years of age and 1 was 7 years of age. All except one contained little or no pigment. The polar spongioblastomata (11 cases) originated in the retina in 6 and in the choroid in 5 cases. These latter tumours appear to occur only in adults, according to the limited information available; all except 2 contained an appreciable amount of pigment. As both nuclear variations and invasions were absent these tumours were presumed to be benign.

The criteria for the choroidal tumours are on a different basis. The choroid forms the outer covering of the retina, a neural structure, and it might be expected that it would give rise to tumours similar to the perineural fibroblastoma or neurofibroma, but no tumours of this type were found in the present series; the majority, however, showed a specific differentiation into choroidal structures.

Besides the melanomata the tumours of the choroid are essentially of 2 groups and these are here classified either as polar spongioblastomata or as sarcomata. Neither show the characteristics of the perineural fibroblastoma or neurofibroma. The spongioblastoma of the choroid not uncommonly shows palisading of the fusiform cells very similar to that seen in the perineural fibroblastoma; in the spongioblastoma, however, there is no relationship between the neoplastic tissue and any form of connective tissue. In direct contrast these choroidal tumours invariably show neuro-epithelial features. If these tumours are entirely within the choroid the question arises as to how they could be neuro-epithelial rather than connective tissue in origin. There is the possibility that the sectioned tissue does not represent the part where the choroidal and the retinal neoplastic tissue are continuous. In other words, it is just possible that here we are dealing with tumours of the retina and that the section merely

shows tissue that had invaded the choroid. The other possibility is that these tumours have arisen from embryonic rests.

The sarcomata of the choroid are instructive in that there is in these tumours a definite attempt to reproduce the hemangiomatous appearance of the choroid, a point that suggests that these tumours are specific tumours of the choroid and not sarcomata of the general type. According to this view, these tumours of the choroid may be classed as the less differentiated (round cell), and the more differentiated (spindle cell) types of malignant tumours of the choroid; the degree of malignancy would probably vary accordingly. In the past the more obvious tumours of this type in which there has been choroid differentiation, have been classed as haemangiomata or as haemangio-endotheliomata of the choroid, either benign or malignant as the case may be. The majority, however, have been considered as malignant melanomata, and largely because they contain pigment.

TABLE IV

Comparison of the Amount of Pigment Present in the Various Types of Tumours

TUMOURS OF THE RETINA

Type of Tumour	No. of cases	*Amount of Pigment*						
		0	±	1+	2+	3+	4+	5+
Neurocytoma	2	1	0	1	0	0	0	0
Neuroblastoma	3	1	0	2	0	0	0	0
Polar Spongioblastoma	7	1	1	4	0	0	1	0
Apolar Spongioblastoma (retinoblastoma)	13	7	1	4	0	0	1	0
Neuroepithelioma	7	2	0	2	2	0	1	0
Total	32	12	2	13	2	0	3	0

TUMOURS OF THE CHOROID

Polar spongioblastoma	6	0	0	2	1	1	2	0
Sarcoma								
(1) spindle cell	13	0	0	4	2	4	2	1
(2) round cell	3	0	1	2	0	0	0	0
(3) myxo	1	0	0	1	0	0	0	0
Melanoma	4	0	0	0	1	0	1	2
Total	27	0	1	9	4	5	5	3
Sarcoma ciliary body	1	0	0	0	1	0	0	0

To what extent is the melanotic pigment of significance? Table IV shows that the melanotic pigment was definitely present in 20 of the retinal tumours out of a total of 32 in all. Amongst the choroidal tumours, however, none was without pigment and all but 1 showed more than a trace. In inflammatory disease of the eye it is generally accepted that any disturbance of the choroid is likely to give rise to increased melanin production in the disturbed part. The result of the analysis of Table IV shows that only 5 retinal tumours out of a total of 32 had 2+ or more of pigment, while amongst the choroidal tumours only 10 showed readings below that amount. The following is a more detailed analysis. While none of the choroidal tumours was without pigment yet the retinal group of tumours had 37·5 per cent. that fell into this group. A moderate amount of pigment (2+) or less was present in 84·4 per cent. of the retinal tumours and in 37·1 per cent. of the choroidal tumours. More than a moderate amount (2+) was found in 15·6 per cent. of the retinal tumours and in 62·9 per cent. of those originating in the choroid. This would confirm the opinion already stated that melanotic pigment in itself is not sufficient for the diagnosis of melanoma. It suggests that the choroid has been disturbed; it is present in retinal tumours, but the larger amounts are more likely to be found in the choroidal tumours.

Summary and Conclusions

1. Intra-ocular tumours are essentially of three types:
   (*a*) Retinal tumours of neural origin.
   (*b*) Sarcomata of the choroid showing choroidal differentiation.
   (*c*) Melanomata of the choroid.

2. Retinal tumours can be classified on a developmental basis analogous to the classification of Bailey and Cushing for glial tumours of the central nervous system. Accordingly, they can be divided into, neuro-epitheliomata, apolar spongioblastomata, polar spongioblastomata, neuro-blastomata, and neuro-cytomata.

3. Sarcomata of the choroid although they can be divided morphologically into the round cell and spindle cell types, and an occasional tumour of the mixed group, *e.g.*, myxo-sarcoma, usually show differentiation into recognisable choroidal structures in parts. Hence they should be considered as malignant choroidomata.

4. Melanomata are comparatively few and contain the typical branched melanotic cells.

5. Pigment may be present in many retinal tumours and is therefore not diagnostic of a melanoma.

To Professor S. L. Baker I am grateful for his interest and helpful criticism. I am indebted to Mr. H. C. Taylor of the Department of Pathology, Manchester University, for the photomicrographs.

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RADON TREATMENT OF SECONDARY CARCINOMA OF THE CHOROID.*

Post-Mortem Observations

BY

P. JAMESON EVANS

BIRMINGHAM

IN September, 1937, I reported the use of radon seeds in the treatment of secondary carcinoma of the choroid in a woman 41 years old. The report described the appearance of growth in the sole remaining eye during March, 1936, the other having been removed for the same condition in January, 1936, and its treatment by radon with disappearance of growth for a period of seven months, and the similar treatment of a further metastasis in the same eye in October, 1936.

The subsequent history is of some interest and is briefly stated thus:—

January, 1937. Vision was 6/12 (i) and there was no evidence of the presence of choroidal growth. A severe reaction of the choroid, similar to that already present in the lower half of the eye, appeared and continued. The retina remained flat but, with the passing of time, became infiltrated with choroidal pigment derived from the stippled pigment in the affected area. The whole macular region was involved and it was surprising how central visual acuity could be maintained. However, the patient continued to maintain vision of 6/12 and could thread her own needles with suitable glasses.

In March, 1937, the patient had severe attacks of vomiting,

* Use of Radon in the Treatment of Metastatic Carcinoma of the Choroid. *Brit. Jl. of Ophthal.*, p. 496, September, 1937.

and pain in the left leg and thigh, and was admitted to hospital for a month. During this time she improved, but X-rays showed rarefaction of the bones of the pelvis on the left side. In May, 1937, vision in the right eye was 6/6 part and there was no choroidal tumour. The patient appeared to be better in general health and by July, 1937, had put on 1 stone 6 lbs. in three months. In August, 1937, the right disc seemed a little paler and vision was 6/9 part. The forward migration of choroidal pigment into the retina was becoming very marked. In November vision was 6/12, but weight was two stone better than in April at 9 stone 12 lbs.

In January, 1938, vision had fallen to 6/18, but there was no change in the fundus to account for this and it was thought that retinal degeneration must be slowly increasing. The patient complained at this time of pain in the right arm and X-rays of the spine showed decalcification of the body of the seventh cervical vertebra. In May, 1938, vision had fallen to 6/36, though there was no appreciably visible change in the fundus picture. On July 16th the patient was re-admitted with a left hemiplegia and in a sinking condition, and died on July 25th, 1938, six years after the removal of the primary growth from the breast and two and a half years after the appearance of metastatic choroidal growth. For two years good vision in the sole remaining eye was maintained and the gradual failure of vision during the last six months of life was subsequently traced to an extra-ocular cause. As an example of local treatment this case must be considered to have been successful in so far as it maintained vision in a patient who would otherwise necessarily have been totally blind.

Post-Mortem Observations

General.—Extremely widespread carcinomatosis was found involving almost all the viscera except the kidneys, uterus and the gut.

Central Nervous System.—A small hard scirrhous growth was found on the upper surface of the right optic nerve, involving the optic chiasma and extending towards the floor of the third ventricle (Fig. 2). There was a further mass the size of a marble in the right half of the cerebellum and several masses growing from the falx cerebri in the superior longitudinal fissure, one in this position about the size of a walnut pressing on the medial aspects of the pre-Rolandic gyri. An area of softening of recent origin was seen in the left parietal region close to the superior longitudinal fissure.

Section of the right optic nerve at the position where it passed over the deposit just in front of the chiasma showed a marked

flattening of the nerve, and atrophy of the part in immediate contact with the tumour (Figs. 2 and 3). The latter, however, was well defined and showed well-marked formation of acini; it did not appear to be infiltrating the optic nerve itself though it is possible that some infiltration had occurred along the meningeal

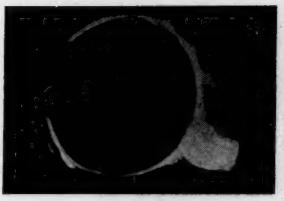


FIG. 1.

Hemisection of right eye.

FIG. 2.

Growth pressing on right optic nerve, gross appearance

FIG. 3.

Growth pressing on right optic nerve microscopical appearance.

sheaths of the nerve. The part of the nerve more distal from the growth was not infiltrated.

The Right Eye was removed, together with the optic nerve throughout its intra-orbital and intra-foraminal length and the whole of the orbital contents.

No growth was found in the extra-ocular contents of the orbit. The eye itself was sectioned after being fixed in 5 per cent. formalin (Fig. 1). The general appearance of a cross section of the eye was not abnormal, but the sclera was unusually

tough on sectioning. The retina was flat and no tumour apparent microscopically. (Fig. 4.) On microscopic section the appearances were of great interest especially as an example of the effect of local radiation. The lens itself was free from opacity; this contrasts strikingly with the frequency of cataract formation following the use of deep X-ray therapy near the eye. The retina was remarkably healthy in appearance though some slight degeneration of the bacillary layer was observed at the posterior pole. It appeared to have suffered little from the absence of a

FIG. 4.

Section of eye: absence of choroidal tumour.

normal choroidal circulation. The choroid in the posterior half of the globe was almost totally absent, only a few degenerated pigment cells remaining. A clear space separating the retina from the sclera indicated the position of the original choroid. Passing forwards, the space became gradually more vascular and behind the pars plana assumed an almost normal appearance.

The sclera showed little, if any, hyaline degeneration as is usually found in fibrous tissues after irradiation. The innermost layers of the sclera showed surprisingly the presence of a fine linear infiltration by carcinoma cells (Fig. 5) which, although showing no tendency to the formation of glandular tumour masses, appeared healthy and did not give the impression of being cells damaged

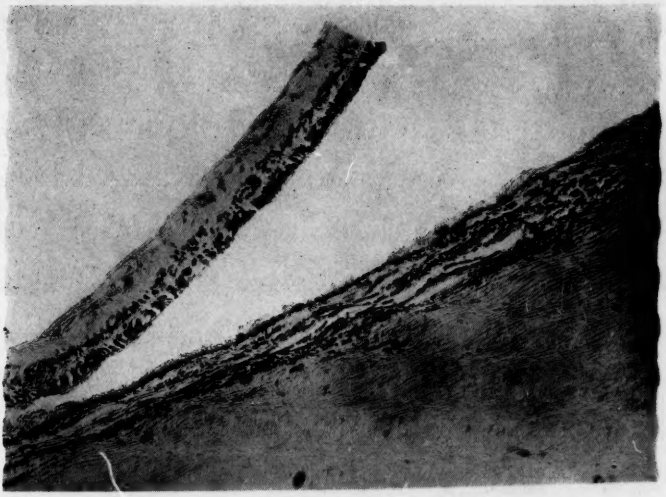


FIG. 5.

Infiltration of sclera by carcinoma cells: the retina fairly normal.

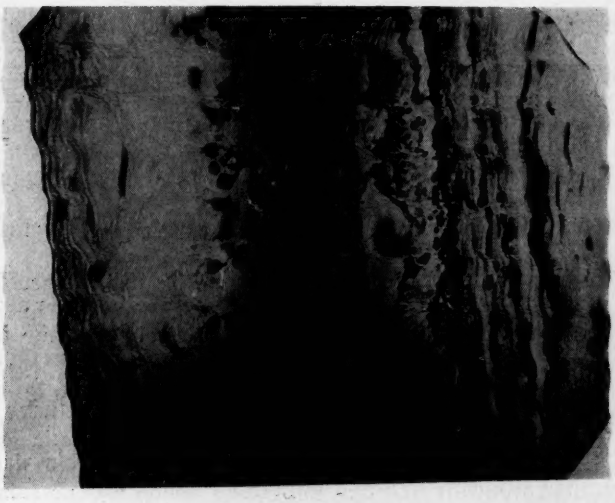


FIG. 6.

Linear infiltration of inner part of sclera by carcinoma cells: absence of choroid.

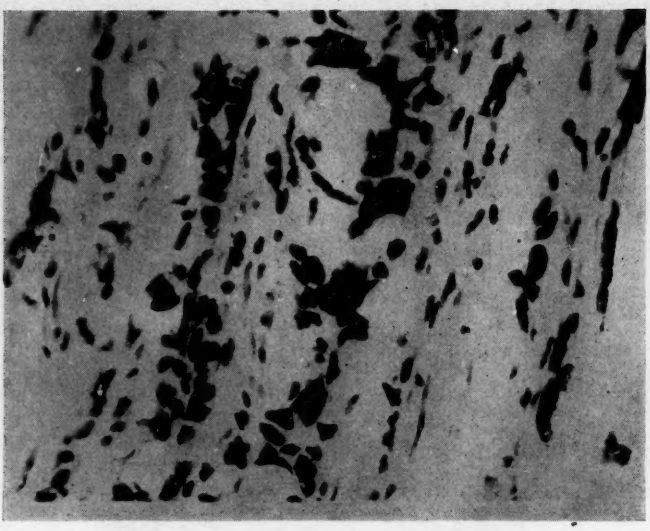


FIG. 7.

High power view of carcinoma cells in sclera.

by radon therapy (Figs. 6 and 7). Whether these cells were quiescent remnants of the choroidal tumour last irradiated some twenty-one months before death, or whether they represent infiltration reaching the eye along the optic nerve sheaths, and derived from the tumour situated above the optic nerve immediately in front of the chiasma, is difficult to say, but pathological opinion is in favour of the latter course. If this be the case it must be presumed that, had the patient survived much longer, further recurrence of metastasis in the eye would have become clinically apparent.

The optic nerve showed a considerable degree of vacuolation and an excess of neuroglial tissue. The degree of optic atrophy was, however, far from complete. No infiltration of the immediately retro-ocular portion of the nerve by growth was found.

In the light of the post-mortem findings the gradual failure of vision during the last six months of life appears to have been due to pressure on the right optic nerve by metastatic growth situated intracranially. It must be confessed that this was not appreciated clinically and that the failing vision was presumed to be the result of defective nutrition of the retina following on the widespread choroidal atrophy at the posterior pole, hence charts of the visual field during this period were not taken. The retina,

however, remained histologically healthy in appearance and visual fields would doubtless have showed a pressure atrophy spreading up from the lower field.

Conclusions

Radon therapy for secondary carcinoma of the choroid, in the case described, proved clinically effective over a period of two years and four months. During the major portion of this period good vision was maintained.

Ultimately failure of vision was due to intracranial complications involving pressure on the optic nerve.

Radon may prove to be of value as a curative form of treatment in an otherwise hopeless condition and should be considered as an alternative to enucleation of the eye.

CONGENITAL FAMILIAL CATARACT WITH CHOLESTERIN DEPOSITS

BY

M. RIAD BEY

CAIRO

THE hereditary factor in many eye diseases has been frequently observed. Of these diseases cataract affords the best example. Nettleship has published details of twenty-two families in which lamellar cataract was found in several generations. The same author and Ogilvie recorded an extraordinary series of discoid post-nuclear cataract in a family called Coppock. Hence the name of Coppock cataract was given to it. On the other hand we frequently find isolated crystals embedded in transparent lenses, chiefly in the peripheral part of the lens under the posterior capsule. This condition according to Pellaton is found in 28 per cent. of clear lenses. Cholesterin crystals are not infrequently found in different types of senile, traumatic and complicated cataracts. Burdon Cooper thinks that cholesterin crystals are extremely common in glycosuria, while tyrosin is the commonest crystal to be found in senile forms.

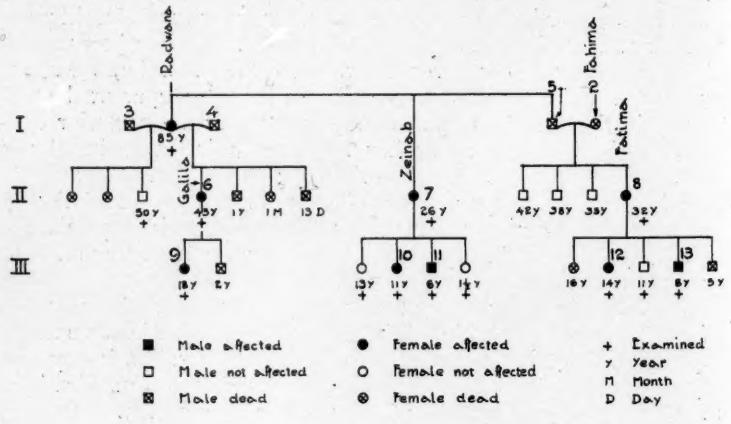
Goulden and other observers have also described the presence of fine crystalline opacities showing green and blue iridescence in thyroid dysfunction, post-operative tetany, Mongolian idiocy and myotonia atrophica. In such cases the crystals are situated chiefly in the anterior and posterior layers of the lens cortex. Doggart reported having seen at Moorfields members of three generations aged twenty years and upwards, in a large family, suffering from

a peculiar type of slowly progressive cataract, showing numerous blue, green and violet dots situated chiefly in the adult nucleus and the superficial layers of the foetal nucleus. Chow recorded a case of cholesterin crystals with cataract in a student, aged 16 years, distributed in the deep layers of the cortex as well as in the nucleus. The crystals diminished in number towards the periphery.

It seems, however, that the presence of crystals in congenital forms of cataract is very rare.

During the annual meeting of the Ophthalmological Society of Egypt for the year 1935 I reported a case of bilateral congenital cataract associated with numerous crystals grouped towards the axial part of the lens. Owing to the difficulty which exists in obtaining an accurate history from our class of patients, the family history of this case was recorded as negative. In May, 1937, a similar case was discovered at the clinic of the Faculty Hospital which presented the same features. This state of affairs induced me to make further inquiries for a reliable history and it was found that the second patient (No. 7, see chart), was a step-sister to the first (No. 6). Further investigations resulted in the discovery of a most interesting pedigree of familial congenital cataract with deposits of cholesterin crystals. I have succeeded in getting into touch with all available members of this pedigree (see chart).

Patients No. 1, 6, 7, 8 were admitted as in-patients at the Faculty Hospital with a view to further study, while the rest were examined at their homes in Giza, Hawamdieh and Cairo districts.



Pedigree Chart

Report on Cases

Past and present history.—The age of the members examined in this pedigree ranges between 6-85 years. Of the 13 cases examined 7 females and 2 males were affected. There is no history of consanguinity in their parents, nor of mental trouble or convulsions. Two step-sisters (1, 2), constitute the material of this report. There is a vague history that their mother complained of some visual defect, most probably cataract. Patient (No. 1) married three times. By her first husband she had three children, one son aged 50 years is still living and not affected. By her second marriage she had four children. The only surviving member, a female (No. 6, the subject of my previous contribution), and her grand-daughter are both affected with this disease. By the third husband, who is at the same time her brother-in-law, being previously married to her step-sister (No. 2), she had one daughter and two grand-children who have inherited this anomaly. Her step-sister (No. 2) had also transmitted this type of cataract to some of her descendents. It is to be observed from the pedigree chart that hereditary influence is strongly marked in the females and that the cataract has been derived from an affected female by continuous descent in three generations.

Investigations.—No constitutional disease was found. No clinical evidence of hypoparathyroidism or hypothyroidism or myotonia atrophica. The presence of cataract and crystal formation was bilateral except in patient (No. 1), who lost her left eye early in childhood probably through purulent ophthalmia. Owing to the similarity of the clinical appearance of the cataract in all the cases examined, one description seems to be quite adequate. With focal examination the cataract appears to be composed of discrete or confluent, nearly circular, opacities of white or grey colour, grouped towards the axial part of the lens in the position occupied by the lines of the lens sutures. They extended in some cases deeply backwards in the lens substance. In other cases the opacities were tube-like in appearance radiating forwards and outwards from the centre. They terminated just short of the lens capsule. Numerous fine shining crystals could be focused scattered in the middle of these opacities. The peripheral part of the lens was quite clear. (See plate.)

The slit-lamp microscope revealed that the space between the anterior capsule and the zone of disjunction was quite clear. In some cases the optical section of a narrow beam localised the opacities and crystals as extending deeply from the anterior cortical zone to the posterior. In others it fell short of the posterior zone of the foetal nucleus.

Cataract with cholesterin deposits.

With the broad beam the opacities appeared either as flattened discs more densely opaque at the periphery than at the centre or like a target of grey and white circles. A few others are mulberry-like and look diaphanous when examined by trans-illumination.

The iridescent crystals are arranged at different angles and in different planes. A few appear to be embedded in the masses of lens opacities. The crystals looked like tiny broken glass fragments, rectangular in shape with a notch at one corner and curiously transparent. They were optically indistinguishable from cholesterin crystals. Neither the opacities nor the crystals were to be seen in the periphery of the lens, but only in its axial portion.

Ida Mann states that the central embryonic nucleus is formed during the first three months of foetal life. The second nuclear layer is formed from the *3rd* to the *8th* month. The next layer is laid down during the last week of foetal life and continues to grow layer after layer till the adult period.

As the opacities in these cases are seen in the central embryonic nucleus, I feel justified in stating that this cataract is congenital in origin. Whatever disturbance caused these opacities to appear

must have acted early in foetal life and have been gradually increasing till a later period. Moreover the similarity of all the cases and the presence of the anomaly in both eyes indicates that the condition is a developmental one.

It is interesting to mention that the percentage of cholesterol in the blood was found to be diminished. It ranged between 114-118 mgr. per cent.

A survey of the available literature shows that no similar pedigree has been reported. No description of such a condition has been recorded in the recent publication of Vogt's atlas, Meesman's or Koby's books on the slit-lamp.

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ANNOTATION

The Problem of the Partially Sighted

The problem of the partially sighted is always a difficult one What standard is fair to all concerned in assessing blindness?

A paper by Sir James Barrett in the *Medical Journal of Australia* of August 13, 1938, deals with this subject. He states that the Royal Victorian Institute for the Blind has, as a matter of abstract justice, paid inmates the difference between what they earn and the basic wage. As he says, "the intention was admirable, but unfortunately human nature has as usual asserted itself, and many people who are not blind have pressed for admittance, evidently seeking a secure position and defined wages. With any slackness the institute would have been overwhelmed and rendered insolvent."

The standard of blindness adopted is that of the British Government. Those unable to count fingers at a range of one metre are "blind"; those with vision up to 3/60, are partially sighted; those with vision between 3/60 and 6/60 are considered in association with the other evidence, such as constricted fields, nystagmus, etc. Patients with vision of better than 6/60 are sometimes admitted to the institute, *e.g.*, cases of retinitis pigmentosa with tubular vision, or cases where blindness is inevitable fairly soon.

In paying those partially sighted the Board is guided by the report of the ophthalmic surgeon.

At the present time in the institute the "blind" and the partially sighted are approximately equal in number.

There appear to be no special classes in Australia for the partially sighted. This is a matter which should be rectified and we hope that the newly founded Ophthalmological Society of Australia will bring the matter to the notice of the government with all the force it can command.

Sir James Barrett states that considerable pressure has at times been brought to bear to secure the admission to the institute of those above the standard, some of whom had excellent sight. His most interesting short note ends with an illuminating table of 28 cases which have applied for admission from 1932 to 1937 and were rejected. Several of these had vision of 6/12 and some 6/6 or even better. One case had 6/6 and normal field in his only eye, the other having been excised. The idea that the Royal Victorian Institute for the Blind is a milch cow adds a disconcerting problem to those who have to administer such charities. It is fortunate for the Institute that such attempts as are outlined here do not succeed and the authorities must be grateful to those upright ophthalmic referees who refuse to certify a person as blind when he or she is not.

ABSTRACTS

I.—THERAPEUTICS AND OPERATIONS

(1) **Gifford, Sanford R. (Chicago).—Physical therapy in ophthalmologic practice.** *Arch. of Ophthal.*, February, 1938.

(1) **Gifford** in this paper, considers the various types of physical therapy which may be applied to the eye. Phototherapy in its general form has a definite value in a person of poor general resistance affected with any form of ocular inflammation, calcium and vitamin D being given at the same time. Local application of ultra-violet light on the other hand is inferior in its effects to "röentgen therapy," and is therefore little used. The application of cold may at times be valuable, particularly in diminishing chemosis and congestion after operations for squint and after simple evisceration of the globe. To apply it, finely chopped ice is placed in a rubber glove, the fingers of which have been tied off. This is covered by a single layer of thin gauze, and allowed to rest against the closed lids during the alternate waking hours for the first three

or four days after operation. Infra-red lamps are useful for applying heat in place of hot bathing, as is diathermy. With regard to the latter, if the temperature inside the eye is raised much above 105° F. there is danger of haemorrhage, a careful technique is therefore required. Chronic irido-cyclitis especially the tuberculous form, and vascular disease of the fundus are found to respond well to local application of diathermy. It is also recommended in severe infections of the lid and orbit and in acute dacryocystitis. General diathermy finds its principal indication in the production of artificial pyrexia though the method is not without danger, and is contra-indicated in elderly or infirm patients particularly if cardio-vascular disease is present. It is of greatest value in gonococcal ophthalmia in the adult, in interstitial keratitis and in syphilis of the central nervous system. X-rays have been used with great success in severe blepharitis. Three doses of 80-90 röentgens are given at intervals of two weeks, a mercury filled glass or a lead shell being placed beneath the lids to protect the eyeball.

F. A. W-N.

(2) **Holmin, N. and Ploman, K. G. (Stockholm).—Thrombosis of central vein of retina treated with heparin.** *Lancet*, March 19, 1938.

(2) **Holmin** and **Ploman** discuss the non-toxicity of heparin, an agent used intravenously for the prevention of thrombosis. They claim that this case of central retinal vein thrombosis is the first in which an already developed thrombosis in man has been treated by heparin.

The description of the ophthalmoscopic findings is as much suggestive of papilloedema and optic neuritis, except for haemorrhages near the periphery at the beginning, which the authors consider characteristic of venous thrombosis. They comment that the obstruction could not have been complete as the symptoms would then have been more serious.

By 3 to 4 daily intravenous injections of heparin 1·66 mg. per kg. body weight for about 10 days the authors suggest that further growth of the thrombus, which was partly occluding the central retinal vein, was prevented, that the fibrin present retracted, the vein dilated and admitted a flow of blood past the thrombus and so circulatory obstruction was reduced. The visual acuity improved from 0·2 to 0·9 in 4 weeks and in 12 weeks from the onset of the trouble it was 1·0 and all retinal haemorrhages had disappeared. The authors attribute the disappearance of the haemorrhages to the fact that they became more liquid under heparin treatment and were thus reabsorbed more easily and rapidly.

They ascribe the favourable course of the case to heparin treatment.

H. B. STALLARD.

(3) **Michie, A. M. and Webster, M. H. (Aberdeen).—Gonococcal ophthalmia treated with 2-p-amino-benzenesulphonamido-pyridine.** *Lancet, August 13, 1938.*

(3) **Michie** and **Webster** report two cases of gonococcal ophthalmia which responded rapidly to M and B 693 tablets, 0·25 g. taken orally twice daily in addition to local treatment of the infected conjunctival sacs.

In case 1 after three days treatment the gonococci and pus cells were absent from smears and swabs taken from the conjunctival sacs and on the sixth day treatment was stopped and there was no further recurrence.

In case 2 treatment with 0·25g. orally began on the evening of July 16 and by 5 a.m. next morning the discharge had almost ceased and by the evening of July 18 all discharge, chemosis and oedema had disappeared, the cornea was clear and on July 19 a smear showed very scanty pus cells but no gonococci and a swab failed to show the presence of any of these micro-organisms.

The authors comment that in 10 previous cases of gonococcal ophthalmia not treated with M and B 693 tablets the average number of days from admission to the first permanent negative smear and culture was 49 and that three of these 10 cases showed some permanent corneal opacity on discharge.

It seems that the reduction of the duration of the ocular phase of this disease may prevent corneal complications in some cases. This treatment appears to be a therapeutic advance worthy of an extended trial.

H. B. STALLARD.

(4) **Meyer, W. L. (Dresden).—Results of the treatment of lid cancroid with Thorium-X-needles. (Ergebnisse der Behandlung der Lidkankroide mit Thorium-X-Stäbchen).** *Klin. Monatsbl. f. Augenheilk.*, Vol. C, p. 321, 1938.

(4) In a series of twenty cases of lid cancroid observed over several years **Meyer** obtained a cure in 18 (90 per cent.) by the use of Thorium-X-needles. The requisite dose is 0·5 millicuries to 1 c.c.m. of indurated tissues. Thorium-X loses its activity rapidly—it sinks to half in three days and is entirely lost at the end of the week.

The article is well illustrated and contains details of treatment.

D. R. CAMPBELL.

(5) **Wegner, W. (Freiburg).—On the operative treatment of a case of injury to the trochlea. (Zur operativen Behandlung der Trochleazertrümmerung).** *Klin. Monatsbl. f. Augenheilk.*, Vol. C, p. 20, 1938.

(5) **Wegner** describes the operative treatment of a case of injury to the trochlear attachment of the superior oblique muscle.

A new pulley is made with a non-absorbable suture (Synthofil). There was a rapid disappearance of diplopia and a good functional result.

D. R. CAMPBELL.

(6) **Thurel, R. (Paris).—Carbolisation of the spheno-palatine ganglion in ophthalmology. (La phénolisation du ganglion sphéno-palatin en ophtalmologie).** *Arch. d'Ophtal.*, Tome 2, p. 506.

(6) Thurel points out that as a result of having obtained relief in various cases of different forms of face-ache by alcoholisation of the trigeminal nerve one has been led on to treat sympathetic neuralgias of the face by carbolisation of the spheno-palatine ganglion itself, as it is the most important and at the same time it is the most easily approachable.

Alcoholisation of the ganglion is the most efficacious method, but as this is not without some danger it is better to content oneself by applying carbolic acid tampons by the intra-nasal method. He points out the numerous ocular troubles that are thought to be of nasal origin, and the difficulty that there is in distinguishing between the anatomical, and the physiological causation. Amongst the conditions he has found to benefit by this treatment are:—Secondary reflex sympathetic neuralgias, periorbital neuritis, whether secondary to injury or to other ocular affections such as episcleritis, iritis or glaucoma, and symptoms like photophobia.

FRANCIS R. HILL.

(7) **Aliquo-Mazzei (Arezzo).—Treatment of rupture of the sclera. (Trattamento delle rotture traumatiche della sclera).** *Boll. d'Ocul.*, February, 1938.

(7) The treatment of rupture of the sclera differs according as the rupture is direct, caused by a perforating wound, or indirect caused by contusion; these latter are comparatively rare accidents; they have certain characters in common; the wound is usually situated in the upper or upper and inner quadrant of the globe and concentric with the cornea; the wound is usually of considerable size and some of the contents of the globe are often forced out of the eye; owing to its elastic nature the overlying conjunctiva is often unbroken; in this case the wound is not infected at the time of injury and may run an aseptic course. The condition of the contents of the globe is the factor of greatest importance in the diagnosis. The lens is almost always extruded and this is a comparatively minor event; if the internal membranes have suffered little damage, the wound may heal and vision may be restored in large part; prolapse of the iris and of the ciliary body and choroid are much more serious; loss of vitreous or copious haemorrhage

may lead to shrinking of the eye and detachment of the retina. Sympathetic ophthalmitis is rare after rupture of the globe, especially if the conjunctiva is intact. Diagnosis of rupture of the globe is usually easy, though subconjunctival haemorrhage may mask it at first. The author advises caution in treatment and prognosis. Even when the conjunctiva is torn, it is not necessary to enucleate at once; the risk of sympathetic does not arise until a month after the injury. The wound in these cases must be cleaned (strong antiseptics are inadvisable) the prolapsed tissue cut off and the edges of the wound sewn up; care must be taken not to increase the prolapse in doing this. Finally a conjunctival flap must be brought over the wound by sliding and secured. **Aliquo-Mazzei** holds that in extensive gaping ruptures operation should be undertaken, even when the conjunctiva is intact. A large conjunctival flap should be made and placed so that the line of suture does not coincide with that of the suture of the globe, and the accident dealt with as in "compound" ruptures. He reports a case in which enucleation had been suggested by a surgeon and refused by the patient, who eventually regained a large part of visual acuity.

HAROLD GRIMSDALE.

(8) **Cerise, L. and Offret, G. (Paris).—Ultimate result of an orbital graft. (Evolution tardive d'une greffe orbitaire).** *Arch. d'Ophtal.*, Tome 2, No. 2, p. 132, 1938.

(8) **Cerise** and **Offret** suggest that the ultimate outcome of orbital operations involving inclusions or the formation of stumps is not as well understood as it ought to be. For one thing it is only the fortunate results that are exhibited. They quote a case where after the enucleation of the eye on account of buphthalmos the resultant disfigurement necessitated an orbital graft and the material used for the inclusion was calf's tendon.

The immediate result was quite satisfactory but after 18 months the patient returned as she could no longer retain the prothesis. The orbital cavity was found to be filled by a cone-shaped sac, apparently in the nature of a cyst. This was evacuated and the lining membrane extirpated, but of the included plug of calf's tendon there remained not a trace. The nature of the best material to employ in such cases is then reviewed and considered along with the accompanying local conditions and the age of the patient. The impression gained is that in young patients, at least, inorganic material is to be preferred.

F. R. HILL.

(9) **Koller, Carl (New York).—Local blood-letting in ophthalmic practice.** *Arch. of Ophthal.*, March, 1938.

(9) **Koller** is of opinion that blood letting is beneficial for a number of ocular conditions in which its value is not fully realised.

In persistent episcleritis, the disease may be disposed of in a large proportion of cases by the following procedure:—Direct the patient to turn the eye inwards if the congestion is temporal or outwards if it is nasal. Hold the lids apart with the fingers, and with a pair of sharp pointed iris scissors, held tangentially to the surface of the eyeball, make a V shaped wound in the conjunctiva and episclera about 7 mm. long. Cocaine should not be used because of its vaso-constricting effect which will diminish the resulting haemorrhage. No bandage is necessary. In iritis and iridocyclitis, especially when dark congestion is present, several such incisions 7-8 mm. from the limbus on the lower half the eyeball will have a favourable influence. Incisions with a sharp scalpel across the conjunctiva of the upper tarsus, and fornix are useful in chronic trachoma with exacerbations and in obstinate forms of subacute conjunctivitis. In recurrent abrasions of the cornea, the author claims that scarification of the conjunctiva followed by brushing the lids lightly with 0·25 per cent. silver nitrate can produce brilliant results.

F. A. W-N.

(10) **Spaeth, E. B. (Philadelphia).—Ptosis and its surgical correction).** *Jl. Amer. Med. Assoc.*, Vol. CIX, No. 23, p. 1889.

(10) **Spaeth** pleads for diversified surgery in the correction of ptosis. There are three muscles of importance to be considered, levator palpebrae superioris, occipito-frontalis with corrugator supercilii and rectus superior. Certain circumstances, when present, indicate definitely the necessity of using one of these, if not exclusively at least to a major degree.

The levator arises by a short tendon which is blended with the underlying origin of the superior rectus. This illustrates the reason for poor levator action in congenital ptosis when combined with poor superior rectus action. The common nerve supply to them is equally relevant. The fibres spread out in a fan-like manner and are inserted in the skin of the upper lid at and below the upper palpebral sulcus (passing through the fibres of the orbicularis), into the superior border of the tarsus, to the superior fornix of the conjunctiva and to the upper border of the margin of the orbital opening.

The frontal part of the occipito-frontalis arises from the epicranial aponeurosis and is inserted into the skin at the level of the eyebrow.

The form of ptosis which is most amenable to surgical treatment is congenital ptosis with an isolated paralysis of the levator; while in myasthenia gravis there is an absolute contra-indication to operation, for the degree of ptosis is always changing.

The principles of operation depend on the shortening of the eyelid itself, advancement of the levator, replacement of the levator by the occipito-frontalis, and, last the utilization of the superior rectus.

In very moderate degrees of ptosis, and in special cases, tarsectomy may be a satisfactory procedure.

Unilateral ptosis must be handled quite differently from bilateral ptosis; also the ptosis of infants must be treated quite differently from that of adults. The ptosis of infants should be corrected as soon as the infant begins to walk, or he will learn to throw his head back and develop a spinal curvature, owing to hyperextension of the head, neck and spine. Crutch glasses may be used here as a stop-gap, until the fourth or fifth year of the child's life.

Success in the employment of Motais's operation depends on the integrity of the superior rectus, but in many cases this muscle has a defective action. In any case it is absurd to suppose that the tarsus-superior rectus adhesion functions as a muscle, if this operation is carried out according to Motais's direction; (this does not apply to the modification described by Young, *Brit. Jl. of Ophthal.*, June, 1924, p. 272).

The utilization of the occipito-frontalis may be a good procedure, but in unilateral cases may result in a peculiar facial grimace.

If there is any action at all in the levator it is probable that advancement of this muscle, combined with tarsectomy, affords relief in the greatest number of cases.

The statement that "all who have had much experience in this branch of ophthalmic surgery will agree with the results of ptosis operations, taken all in all, are far from brilliant" is unfortunately true.

The author gives a resumé of the various procedures which he considers applicable to different ages and different conditions.

A. F. MACCALLAN.

(11) **Barkan, Otto (San Francisco).—Technique of Goniotomy.** *Arch. of Ophthal.*, February, 1938.

(11) Goniotomy, as its name implies, consists in opening up the angle of the anterior chamber of the eye so as to allow the aqueous to drain directly into Schlemm's canal. The type of case suited to this operation is that which **Barkan** has called type 1 of chronic glaucoma in which the depth of the anterior chamber is normal and its angle open. Cases with a shallow anterior chamber should be treated by other methods.

The operation is performed under local anaesthesia by instillation of 1 per cent. pontocaine hydrochloride. A special type of contact lens is used which enables the surgeon when looking from the temporal side of the eye, to obtain a clear view of the angle of the anterior chamber on its opposite (nasal) side. After the lens has been inserted, the space between its posterior surface and the cornea is filled with normal saline, introduced by a syringe

and lacrymal canula. The lens is held on the eye with a "bident" by an assistant, and the surgeon passes the goniotomy knife (which rather resembles a Ziegler needle) from just behind the temporal limbus across the anterior chamber so that its point engages the fibres of the pectinate ligament on the nasal side of the eye. By depressing or raising the handle of the knife it is possible to divide the inner wall of Schlemm's canal over at least a quarter of its circumference. The knife is then removed without loss of aqueous and the operation is completed. Technique is important for a successful result, and details of this are given by the author. Up to date he has operated upon 25 eyes, with good results provided that the incision opened at least a quarter of the circumference of Schlemm's canal. Details of these cases are to be published later.

F. A. W-N.

(12) **Nakamura, B. and Uchida, Y. (Osaka).—On the prophylactic treatment of sympathetic ophthalmitis. (Zur prophylaktischen Behandlung der sympathischen Ophthalmie).** *Arch. f. Ophthal.* Vol. CXXXVII, p. 233.

(12) In a previously published work **Nakamura** had recommended the use of a tuberculous vaccine in sympathetic ophthalmitis before the enucleation of the injured eye, especially where it still retained more or less useful vision.

Subsequent experience of this treatment has convinced him that it is preferable to all others as a prophylactic as well as a therapeutic measure.

Nakamura and **Uchida** here record in tabular form a series of 95 cases of perforating wound of the eye, which had been submitted to prophylactic treatment with tuberculous vaccine and calcium iodide because of the intense inflammation two weeks after the injury, the more or less serious injury of the ciliary body, the perforating wound with subsequent iridocyclitis, the sympathetic irritation of the other eye and the tuberculin allergy that they showed; in none of these did sympathetic ophthalmitis supervene.

Another table is given with details of 139 cases of perforating injury, in which this prophylactic treatment was not employed; the symptoms after the injury in these were comparatively mild, yet eight developed sympathetic ophthalmitis.

The method of treatment employed was the same as previously used for tuberculin affections of the eye, and consisted of a weekly subcutaneous injection of 1 c.c. of tuberculous vaccine No. 1 (vide *Archiv. of Ophthal.*, Vol. X, 1933, pp. 161 and 423, T.S.) and every second day an intramuscular injection of 3 c.c. of 3 per cent. calcium iodide.

THOS. SNOWBALL.

BOOK NOTICES

La Tonoscopia Retinica. By F. ORZOLESI and N. CASSUTO, with a preface by BARDELLI. Pp. 187, with 8 Plates and one Illustration in the Text. Florence: Bemporad and Figlio. 1938. Price, L.25.

The importance of a complete knowledge of the physiology of the retinal vessels, as an indication of the condition of the vessels within the skull, can hardly be exaggerated. In no other region of the body can the blood vessels be observed with the accuracy which is possible here. Since the eye is, in development, an outgrowth of the brain, and its circulatory system is a part of the cerebral (which cannot be observed directly), evidence gained from the retina may be of great assistance to the neurologist. For this reason many researches have been undertaken to discover accurately the pressure in the various parts of the retinal circulation; it is generally held that the ophthalmodynamometer of Bailliart is the instrument which is most suitable for clinical examination. It consists of a spring which is compressed by a rod whose end is applied to the eye: the force exercised is recorded by a hand on a graduated circle. In order that blood should flow through the eye, it is necessary that the pressure must in all parts of the course, be greater at some time than the intra-ocular pressure. It has been known for a long time that in aortic insufficiency, when the diastolic pressure is low, a spontaneous pulsation may be observed in the retinal artery; Bailliart assumes that when pressure on the globe is followed by a visible pulse in the artery, the intra-ocular pressure is equal to the diastolic; when, after further increase of pressure all pulsations disappear, the systolic pressure is found.

Many other forms of instruments are described by the authors but none is so suitable in their opinion for clinical study as this of Bailliart; many are complicated and elaborate; the measurements as taken by them, agree for the most part fairly with those of Bailliart.

He does not claim accuracy for his results; several factors, as for example, the resistance of the vessel wall are not considered. Fritz has suggested that the difference in pressure required to produce pulsation and to occasion complete collapse of the artery during diastole, is the measure of the elasticity of the wall. Though the arterial pressure may be said to be known fairly accurately, much less is known of the pressure in the capillaries and veins; from the frequent presence of a spontaneous venous pulse, it may be assumed that the minimum pressure in the veins is very little greater than the intra-ocular pressure; the pressure in the capillaries must be higher than this.

An important point to be considered is the relation of the pressure in the retinal artery to that of the general arterial system. Bailliart

holds that the diastolic PAR is normally half that of the brachial. He holds that when, in glaucoma, the intra-ocular pressure is equal to the diastolic PAR, the danger of an acute attack is imminent as a corollary of this, it would seem that high arterial tension is a safeguard in glaucoma and he quotes cases in which, the brachial pressure being high, the eye sustained high intra-ocular pressure for years without loss of acuity.

In certain morbid states the normal relation is impaired. When the diastolic PAR is greater than half the diastolic pressure in the brachial, Bailliart regards it as indicating a probable increase in intracranial pressure and the condition is known by his name, as "Bailliart's symptom."

Having dealt at some length with the physiological problems, the authors pass on to consider the ways in which the arterial pressure may be modified; separate chapters deal with the changes produced by drugs, and in ocular and general diseases. A full bibliography covering some thirty pages follows. A summary of contents but no index ends the monograph.

Apart from the "corrigenda" on p. 186 we have noted only two or three misprints; one on p. 51 near the bottom leaves the sense in doubt; the remainder are literal only.

The study was well worth doing. The authors are to be congratulated on a work that seems to contain all that is known on this important subject.

Handbuch der Erbkrankheiten. Edited by A. GÜTT. Band 5. Erbleiden des Auges. Pp. XI +310, with 221 Illustrations. Leipzig: Georg Thieme. 1938. Price, R.M. 24.

This is the second volume that has appeared of a planned series of six to cover hereditary diseases in general, this particular volume being devoted to ophthalmology. The scope of the book is explained in the preface by the editor, who is a medical officer of the German Home Office, as an attempt to bring together the relevant scientific knowledge to help in the application of the German laws on eugenics. Purely academic material is therefore not included as the volume aims at being a practical treatise, though it is also stated that it is necessary to have a sound scientific background to apply these laws intelligently. The text of the book which has been contributed by Bücklers, Clausen, Fleischer, Harms, Jess, Löhlein and Wegner reflects the limits imposed to a rather marked degree. Some sections are useful expositions of the state of knowledge on the particular subject with the references to the practical application to eugenic laws as a mere appendage, almost of an apologetic type. Other sections are essentially a medico-legal discussion with the ophthalmological part reduced to a minimum. With a non-inclusive compilation of this type, the omissions as well as the inclusions are

open to criticism, but on the whole the authors have done their work satisfactorily under difficult conditions. The medico-legal discussions with their reference to actual cases before the courts, illustrate the difficulties in applying the present knowledge of heredity on a wholesale scale and are only of indirect interest to the English reader. The ophthalmological text contains little that has not been covered more fully in the excellent monographs by Waardenburg and Franceschetti. Their most valuable feature is that they bring the references to the literature up to date, the two standard monographs having been published about six years ago. The small section on hereditary corneal lesions by Professor Bücklers deserves special mention as introducing a new classification in this difficult subject, and is presumably based on the larger monograph by this author that has been announced as a forthcoming supplement to the *Klin. Monatsbl. f. Augenheilk.*

An Introduction to Clinical Perimetry. By H. M. TRAQUAIR, M.D., F.R.C.S.(Ed.). Third Edition. Pp. 320, Figures 227. London: Henry Kimpton. 1938. Price, 30/-.

The text of the 3rd edition of this admirable work has been carefully revised, re-arranged and amplified. Certain anatomical descriptions have been brought up-to-date with the result of recent researches. Forty-eight new illustrations and 63 new references have been added.

This book has long been of established value to the neurologist, neuro-surgeon, ophthalmologist and physicians interested in the clinical application of perimetry.

The author advocates the study of simple principles for field testing and states that these lead to more satisfactory results than elaborate procedures with complicated apparatus. He states that the value of quantitive perimetry for the purpose of diagnosis and research is becoming increasingly appreciated. He is careful to emphasize the fact that in many instances visual field changes form only a part of the evidence on which conclusions are to be based. Throughout this work the reader is struck by the author's scientific approach in the assessment of clinical data.

This monograph is well balanced, interesting and of considerable value to physiological research workers and clinicians alike. The illustrations and production are excellent.

Les Etats de la Refraction Oculaire Chez les Palenegrides au Gabon, Afrique Equatoriale Française. By STIG. HOLM. Pp. 299. *Acta Ophthalmologica.* Supplement XIII. Copenhagen. 1937.

HOLM had the opportunity of investigating the study of refractive errors—or refractive variations as he rightly calls them—in a

primitive negroid race in French Africa. His investigation is of importance as there are no reliable data concerning refractive variations in primitive races, a subject that has given rise to much speculation and is of theoretical importance. His study has covered examination of 2,377 eyes in illiterates and 1,339 eyes in patients who could read and had engaged in some type of close work. Attention was paid to the necessary statistical evaluation of these data and his conclusions in broad outline are that there was no tangible difference between the two groups, and that extreme variations were very exceptional, practically all the cases falling between emmetropia and 2 D. of hypermetropia. Plotted out, these findings gave an almost theoretically perfect curve of variation, such as is postulated by Steiger's theory. Holm also draws attention to the fact that the so-called myopic crescents, as also senile crescents, were not observed. He could find no evidence for the anthropometric theory of myopia which looks upon the elongated eye as an adaptation to the structure of the orbit; nor could he find any support for the view that infections cause myopia, for infective conditions were very common in this group. He holds that his study supports those who regard high myopia as a distinct pathological entity, of which a few examples were observed in his group. A point of some interest in his study was the practically complete absence of astigmatism in the whole of his series. Anisometropia was also almost completely absent.

Zur Frage des späteren Schicksals der Phlyktanulosepatienten mit besonderer Berücksichtigung der Tuberkulose. By AARNI AJO. Pp. 159. *Acta Ophthalmologica.* Supplement XV. Copenhagen. 1938.

AJO reviews the literature on the aetiology of phlyctenular ophthalmia and gives an analysis of 449 cases treated at the University Clinic in Helsinki between 1912 and 1927, paying special attention to the question of the ultimate fate of children with phlyctenulosis. He was able to obtain information on 364 such cases and found that 55 or 15 per cent. had died from various causes, death being due in 35 cases (9·56) to tuberculosis, an incidence that is twice as heavy as that for the general population of the corresponding age groups. He was not able to examine all the survivors. Out of 100 with whom he established contact, 50 were completely free from any symptoms, 39 had tuberculous lesions, 5 had lesions suspicious of tuberculosis and 6 were the subjects of other pathological conditions. Seven of the 39 had pulmonary tuberculosis, which on an average developed 10 years after the phlyctenular lesion had first been noted. He concludes that phlyctenulosis does not protect against the subsequent development of tuberculosis—as has been suggested by some—and that phlyctenular ophthalmia is the manifestation of tuberculous infection.

Clinical Investigations of the Inflammatory Orbital Affections. By HOLGER EHLERS. Pp. 123. *Acta Ophthalmologica.* Supplement XII. Copenhagen. 1937.

To the difficult subject of acute inflammatory swellings of the orbit Ehlers has contributed a careful study based on 36 personal cases. Following the teaching of van Rossen and more particularly of Rönne he draws a clear distinction between orbital oedema, subperiosteal (or preseptal) abscess and abscess of the orbit. The first condition gives no localised signs, the second is a localised abscess tending to burst spontaneously, the third is a serious affection which is sufficiently well known to need no further description. Conservative treatment is indicated in orbital oedema, a limited incision in the second, whilst the third calls for more drastic measures. From a study of his cases and analyses made of the primary scources of the infection, special stress laid upon sinusitis in children with acute infectious fevers as a cause of orbital oedema. The monograph also discusses the secondary orbital infection seen in cavernous sinus thrombosis.

Allergische Augenerkrankungen. By Professor ARNOLD LOWENSTEIN. Pp. 72. (*Beiheft No. 26 of Zeitschr. f. Augenheilk.*) Basle: S. Karger. 1938. R.M. 4·60.

PROFESSOR LÖWENSTEIN'S monograph gives a concise review of the theory of allergy as applied to ophthalmology and deals in detail with various ocular reactions which are recognised as allergic or are thought to be such. The scope of this monograph includes discussions on experimental findings such as anaphylactic keratitis and its relationship to interstitial keratitis. A critical attitude is maintained throughout and valuable suggestions emerge throughout the text. Points of interest are the possible allergic basis of familiar corneal dystrophies as also the possibility of the chalazion being an autogenous allergic reaction. This useful review covers much the same ground as the English monograph by Alan Woods on Allergy and Immunity in Ophthalmology.

Zur pathologischen Anatomie und Therapie des Trachoms nach histologischer untersuchung des Tarsus und seiner Bindehaut. By Professor ARTH. BIRCH-HIRSCHFELD. Forty-two pages with 18 illustrations. (Beiheft No. 27 of *Zeitschr. f. Augenheilk.*) Basle: S. Karger. 1938. R.M. 4·10.

BIRCH-HIRSCHFELD who from his great experience in trachoma in Königsberg can speak authoritatively on this subject, has devoted this monograph to a histological study of 153 excised tarsal plates in trachoma. He gives a detailed description of the findings in the conjunctival epithelium, sub-epithelial tissue, pretarsal tissue and

the tarsus itself. He draws particular attention to the cell infiltration around the follicles and holds that too much attention has been given to the follicle as against this infiltration which is the basis of not only scar formation but of necrosis and other histological complications. Treatment should, therefore, aim at reaching these reactions, but where these reactions are situated deeply medication is more difficult, and mechanical, as also surgical, interference finds a new justification.

Das Trachom. (Ergänzungsband). By Professor A. PETERS. *Eighty-five pages.* Basle: S. Karger. 1938. R.M. 4·20.

The present volume is a supplement to the exhaustive treatise on trachoma published by Peters in 1935, and deals with the literature on the subject that has appeared since November, 1934, to February, 1938. It is of the same inclusive character as the original book and like it is essentially of the encyclopaedic type rather than a critical survey. Nothing seems to have escaped the author and it is no fault of his that the section on therapeutics is almost an international pharmacopoea. Like the original volume this supplement is invaluable to the student of the subject.

Die Verätzungen des Auges. By OSKAR THIES. Pp. 103 with 60 *Illustrations. (Beiheft No. 2 of Klin. Monatsbl. f. Augenheilk.)* Stuttgart: Ferdinand Enke. 1938. R.M. 4·60.

Following a general discussion on the various factors and reagents causing ocular burns the author emphasizes the advantages of operative procedures particularly in the more severe injuries. As far as the skin is concerned the general procedures applicable to pedicle grafting are discussed. As for the eye the values of mucous membrane grafting are indicated. Of particular interest is his adherence to the teaching of Denig as to the advisability of early excision of damaged conjunctiva and its replacement by mucous membrane grafting. His advocacy is supported by impressive case results and he fully subscribes to Denig's dictum that the only correct treatment of caustic burns of the eye is immediate grafting.

CORRESPONDENCE

ANTEPOSITIO CONJUNCTIVAE FORNICIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In the *Brit. Jl. of Ophthal.*, October, 1938, p. 636, Dr. Shimkin published a reflection on my re-claimed priority of

what he calls "Antepositio Conjunctivae Fornicis," denying this priority.

Dr. Shimkin only quotes my short note from the *Klin. Monatsbl. f. Augenheilk.*, Vol. LXVIII, p. 617, 1922, in which I simply extended my method—originally devised for trachoma—to cases of severe spring catarrh; but he does not refer to my former paper, which appeared in *Arch. f. Ophthal.*, Vol. CV, p. 542. Anybody who has read *this* paper, entitled, "Kaustische Resektion der trachomatösen Tarsalbindehaut," will agree with me that this is the same operation as "Antepositio Conjunctivae Fornicis," with the only difference that I destroy the affected tarsal conjunctiva by the galvanocautery previous to covering the denuded with conjunctiva from the fornix.

Yours respectfully,

Professor Dr. L. LIEBERMANN.

BUDAPEST, *October* 24, 1938.
Veres Palné Str. 34
HUNGARY.

NOTES

Royal Eye Hospital, Research Department

ON November 2, 1938, Mr. L. Vernon Cargill, chairman of the Hospital, opened the reconstituted pathological laboratory and research department of the Royal Eye Hospital.

He recalled the foundation of the Pathological department nearly 50 years ago and pointed with justifiable pride to the good work done there. The new extension should make it easier to continue the work. A new animal house have been installed, the museum has been overhauled, the specimens has been remounted, relabelled and catalogued. Representative specimens have been starred in the catalogue and provided with labels of distinctive colour. The collection of melanomata of the choroid, intra-ocular foreign bodies and eyes with expulsive haemorrhage is very complete. Representative slides and reference books are available in the out-patient department.

Mr. R. H. Rushton demonstrated his new method for the clinical measurement of the axial length of the eye. The dark adapted eye is sensitive to X-rays. By his instrument a beam of X-ray can be moved forward till it impinges on the posterior pole of the eye and

be recognised entoptically by the patient. At the same time the position of the cornea is registered; simple measurement gives the axial length of the eye.

Mr. E. J. Somerset read a paper on the significance of errors of refraction in children with chronic blepharitis, which we hope to publish early in the new year.

A cordial vote of thanks to all concerned terminated the proceedings.

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Addendum

THE footnote to Dr. John V. V. Nicholls's paper on p. 672 should read: "Travers Allan Travelling Fellow, Royal Victoria Hospital, Montreal.

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Ophthalmological Society of the United Kingdom

THE Annual Congress will be held on April 20, 21, 22, 1939, at the Royal Society of Medicine. The subject for discussion is "The Problems of Refraction." It will be opened by Mr. Affleck Greeves, Mr. Charles Goulden and Mr. Harold Levy. The Bowman Lecture will be delivered by Professor Weve on "Diathermy in Ophthalmic Practice." The Annual Dinner will be held on April 20, 1939. Members wishing to read papers or to take part in the discussion should notify Mr. F. W. Law as soon as possible. The International Organization against Trachoma and the International Association for the Prevention of Blindness will hold their annual meetings on April 19, 1939. Members of Congress can attend these meetings.

FUTURE ARRANGEMENTS

1938

December 6.—Midland Ophthalmological Society (Middlemore Lecture), Birmingham and Midland Eye Hospital.

December 9.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

December 16.—North of England Ophthalmological Society, at Leeds.

1939

January 13.—Section of Ophthalmology, Royal Society of Medicine.

January 20.—North of England Ophthalmological Society, Annual Lecture, at Manchester.

January 21.—North of England Ophthalmological Society, Annual Lecture, at Manchester.

February 3.—North of England Ophthalmological Society, at Newcastle-upon-Tyne.

February 3.—Midland Ophthalmological Society, at Oxford Eye Hospital.

February 10.—Section of Ophthalmology, Royal Society of Medicine.

March 3.—North of England Ophthalmological Society, at Liverpool.

March 10.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

March 31.—North of England Ophthalmological Society, at Sheffield. (Joint Meeting with the Midland Ophthalmological Society.)

April 20-22.—Ophthalmological Society of the United Kingdom, Annual Congress, at the Royal Society of Medicine.

May 19.—Midland Ophthalmological Society, at Gloucester Royal Infirmary and Eye Institution.

June 9.—Section of Ophthalmology, Royal Society of Medicine. (Annual.)

October 6.—Midland Opthalmological Society, at Birmingham and Midland Eye Hospital. (Annual.)

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *October, 1938.*

BRÜCKNER. Physiologic and clinical ophthalmologic problems in relation to individual variability.

THYGESON and RICHARDS. Nature of the filtrable agent of trachoma.

SUDRANSKI. An evaluation of homatropine-benzedrine cycloplegia.

PARKER. A cataract operation to reduce the incidence of prolapse of the iris.

OGLE. Induced size effect: 1. A new phenomenon in binocular space perception associated with the relative sizes of the images of the two eyes.

SEECH and COOPER. Experimental iontophoresis of rabbits' corneas: report of two cases of corneal dystrophy with treatment by ionic medication.

CHANDLER Inferior iridotomy in operations for cataract on eyes with posterior synechiae or pupillary membrane: value of operation.

GUNDERSEN. Results of autotransplantation of cornea into anterior chamber: their significance regarding corneal nutrition.

CASE. An intern's experience with the Verhoeff method of cataract extraction.

American Journal of Ophthalmology. *October, 1938.*

MEYER, SMYTH and GALLARDO. Nature of the ocular fluids.

BORLEY and TAINTER. Effect of dinitrophenol on lactose cataracts.

BARKAN. Glaucoma.

GOLDBACH. Kayser-Fleischer ring—Wilson's disease.

BIELSCHOWSKY. Lectures on motor anomalies.
JULIANELLE, HARRISON and LANGE. Studies on inclusion blennorrhea.
ALLEN. Routine in eye examinations.
HOBSON. Epidemic superficial punctate keratitis.
BINKLEY and MOTTO. Primary epibulbar epithelioma.
SOUTHGATE. A treatment of chronic dacryocystitis.

Annales d'Oculistique. *October, 1938.*

HARTMANN and GUILLAUMAT. Aspect of the fundus in intra-cranial tumours. Statistical studies.
CHARAMIS. Van der Hoeve's syndrome of blue sclerotics.

Archiv für Ophthalmologie. *September, 1938.*

TSCHERMAK-SEYSENEGG. Studies on neutral light tests.
TSCHERMAK-SEYSENEGG. A double slit system with constant wave lengths of mixed spectral lights.
HAASS. Photokinetic theory of skiascopy.
BAKKER. Sodium light in ophthalmology as a help for the unmasking of malingerers.
BAKKER. Contribution to the knowledge of remarkable pupillary movements.
WECKERT. Biology in ophthalmology.
CHOJNACKI. Experimental and clinical studies of dentogenous localized disease.
ZEISS. Changes in the outer lens cortex through the effects of ultra sound waves.

Zeitschrift für Augenheilkunde. *September, 1938.*

PANTASATOS. The intrascleral ciliary nerve loops of the retro-bulbar section.
KURZ. Ocular changes in lupus erythematodes.
HOFFMANN-RÖTZEL. The erythropsia of aphakia.

Klinische Monatsblätter für Augenheilkunde. *September, 1938.*

WÖLFFLIN. Transient alterations of lens opacities from irradiation by means of different wave lengths.
SCHEYHING. Experimental researches on the efficacy of short waves on normal and infected rabbits' eyes.
POSTIC. Significance of Weil-Felix's serological reactions in trachoma.
PASCHEFF. The origin of folliculoma and follicles after Denig's transplantation.
MORETTI. Contribution to the surgery of trachomatous corneal pannus.
MIRIC. A simple operative procedure for entropion and trichiasis following trachoma
FAZAKAS. Acrothecium hominis olah causing Meibomian conjunctivitis.
MEYER. Contribution to the syphilitic diseases of the eyes.
KURZ. Orbital blood cysts.
LUZSA. The influence of enucleation on the formation of the optic nerve canals.

October, 1938.

HEINSIUS. The simple uncomplicated form of congenital total colour blindness.
FREUSBERG. Semi-homonymous papilloedema with partial optic atrophy after a unilateral tract lesion.
SAGHER. Unfamiliar juvenile corneal dystrophy.
HARTMANN. The question of ocular injuries through sulphuretted hydrogen.
DÖHMEN. Bacteriological and chemical researches on the prevention and treatment of ocular infections.

VOGT. Cataract discussion in Heidelberg.
WAGNER, RICHNER and KARBACHER. Preliminary results of the treatment of senile cataract with vitamin B2 (Lactoflavin) administration. Control with slit-lamp microscopy.
BRAUN. The Chaoulsche fractional concentration of X-rays in near irradiation for malignant tumours in the lid.

Bollettino d'Oculistica. *August, 1938.*

BENCINI. Ocular behaviour of rabbits vaccinated against tuberculosis, the tubercular antigen being introduced into the anterior chamber.
MOTOLESE. Bilateral paralysis of the right externus muscle associated with papilloedema from novocain rachianaesthesia.
CASSUTO. The behaviour of the retinal arterial pressure after blood letting.
GRANCINI. An exceptional case of severe ulcus rodens corneae in a young subject, successive manifestations and symmetrical also in the other eye.
NASTRI. Therapy with anatasis thread in staphylococcal infection of the lid.
SCARDAPANE. Severe amblyopia following metrorrhagia in haemophilia.
BOZZOLI. Simple method for localization of the hole in retinal detachment.

September, 1938.

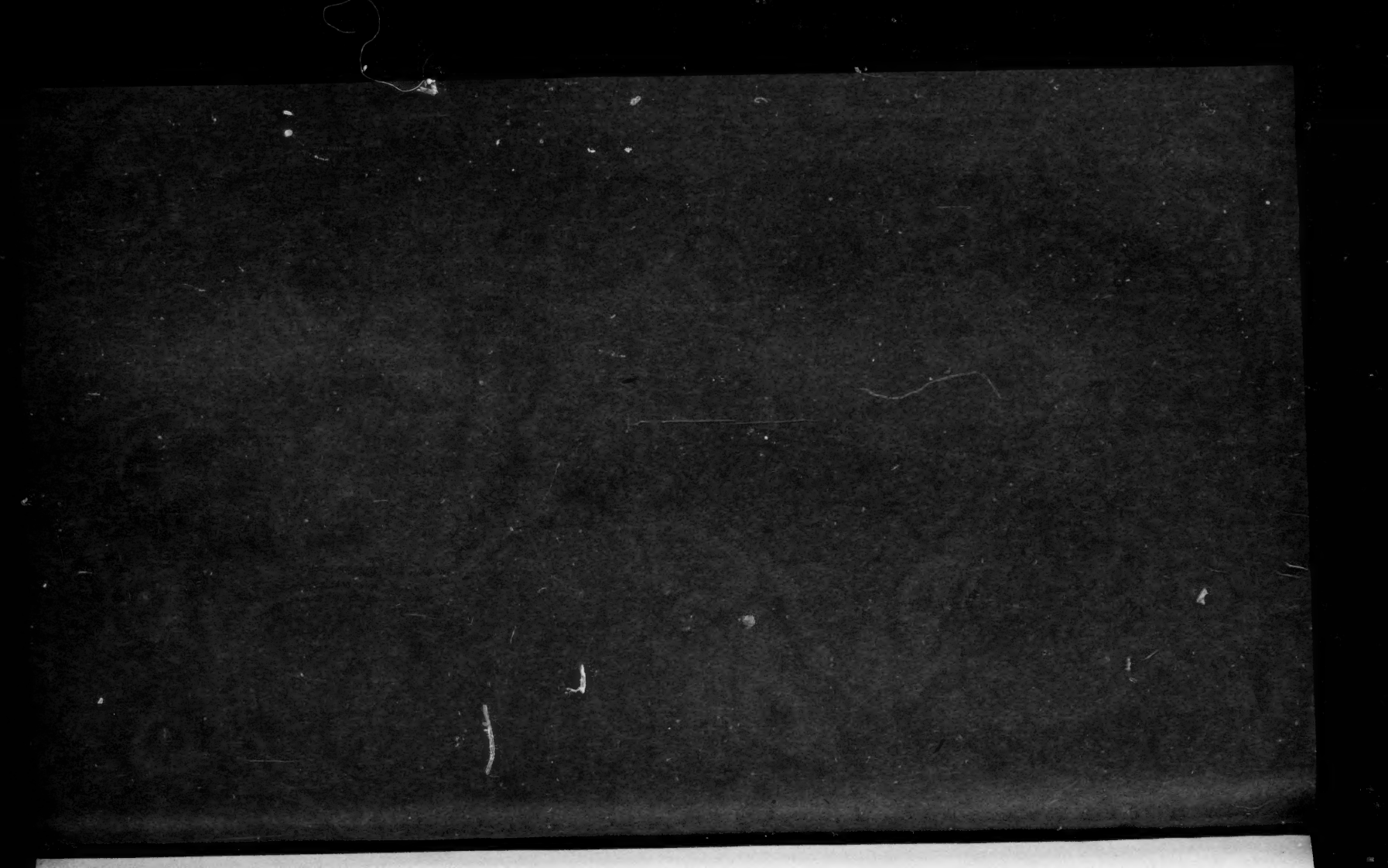
BENCINI. Bipolar electrolysis in the cure of retinal detachment.
BERARDI-MOTOLESE. Hereditary familial corneal dystrophy with recurrent dysepithelisation.
BIETTI. Spectrographic investigation for the presence of metal in the conjunctiva.
CIOTOLA. Clinical research on the behaviour of the ocular tension in hypoglycaemic coma.
SCARDACCIONE. Researches on the action of the melanin producing hormone on the time of dark adaptation and on the threshold of light excitation in the normal individual and in retinitis pigmentosa
BORIONI. Folliculin in the treatment of gonococcal conjunctivitis.
DI BARI. Partial amblyopia in the astigmatic eye revealed and measured by means of Dr. Di Bari's revolving metric quadrant.

Revista Oto-Neuro-Oftalmologica. *July-August, 1938.*

VERCELLI. An undescribed variety of a familial disease: retinitis pigmentosa and myoclonic epilepsy.
BOZZI. Clinical contribution to the study of tumours of the 4th ventricle.
MARSIGLI. Adie's syndrome.

Arquivos Brasileiros de Oftalmologia. *August, 1938.*

VALLE. Hoffman's "Choroidite leprosa precoce."
SILVA. Ocular disturbances after meningitis.
URIOSTE. Treatment of corneal ulcers.

Klinika Oczna.

KARELUS. The physical and biological limit of the sense of light.
WILCZEK. von Hippel-Lindau's disease.
MORAWSKA. The difference between the refraction on the optic disc and on the yellow spot measured by means of the skiascope.
MADROSZKIEWICZ. New keratoscope adapted to Simon's electric ophthalmoscope.
MIESES-REIF. Pigment content in the hair and iris.